Взаимоотношения клеток разных организмов

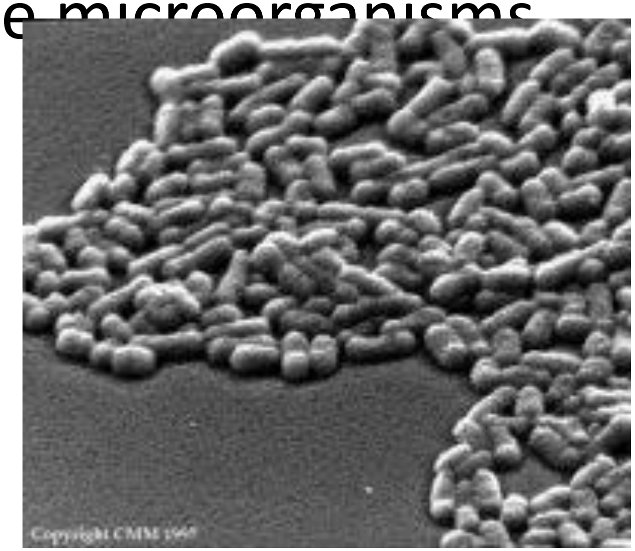
Classification of population interaction

	EFFECT OF INTERACTION	
NAME OF INTERACTION	**POPULATION A**	**POPULATION B**
Neutralism	0	0
Commensalism	0	+
Synergism	+	+
Mutualism	+	+
Competition	-	-
Amensalism	0 or +	-
Parasitism	+	-
Predation	+	-

0, No effect; +, positive effect; -, negative effect.

Mutualism

- in this type of relationship, both partners benefit
  - *E. coli* synthesizes vitamin K in the intestine
  - in exchange the large intestine provides nutrients necessary for survival of the microorganisms



E. coli

Animal–Microbia Symbioses

- The Rumen and Ruminant Animals
- Hydrothermal Vent Microbial Ecosystems
- Squid-*Alvibrio* Symbiosis

Diagram of the Rumen and Gastrointestinal System of a Cow

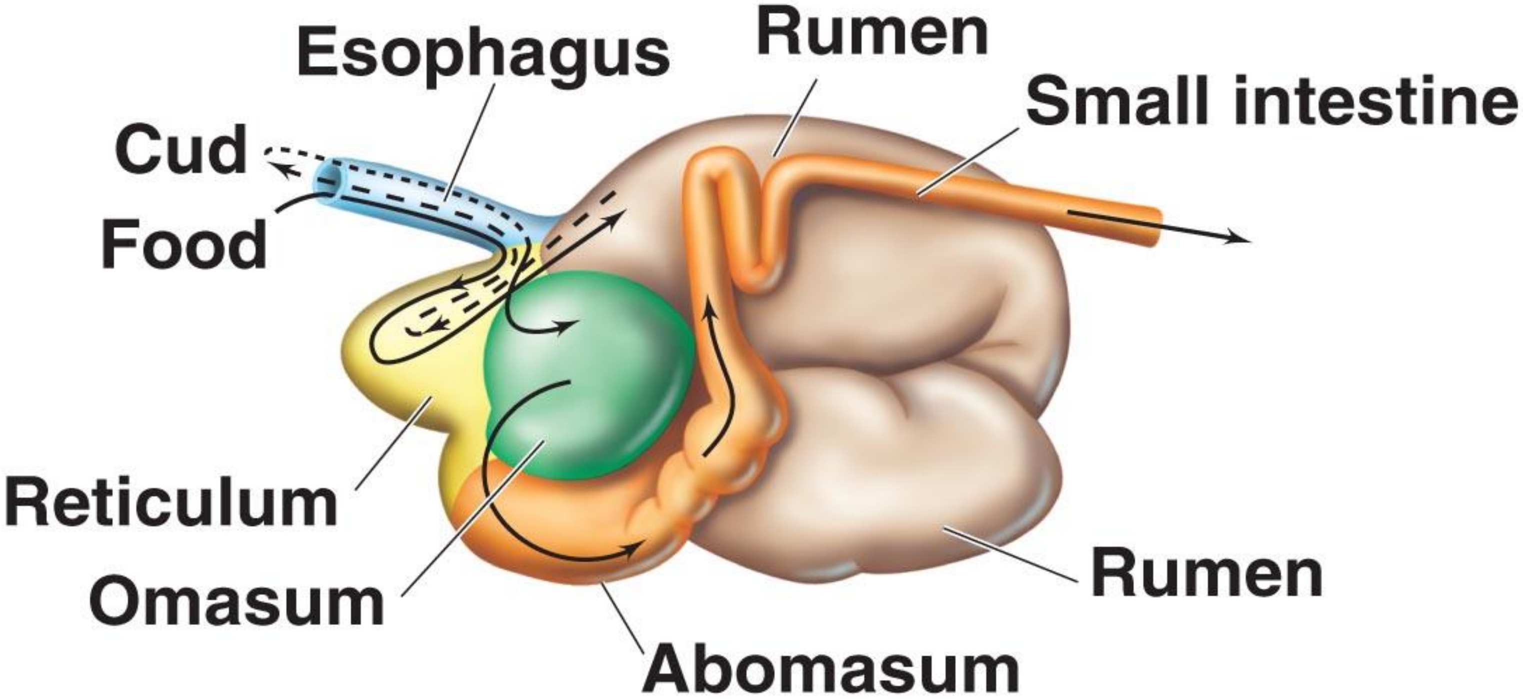


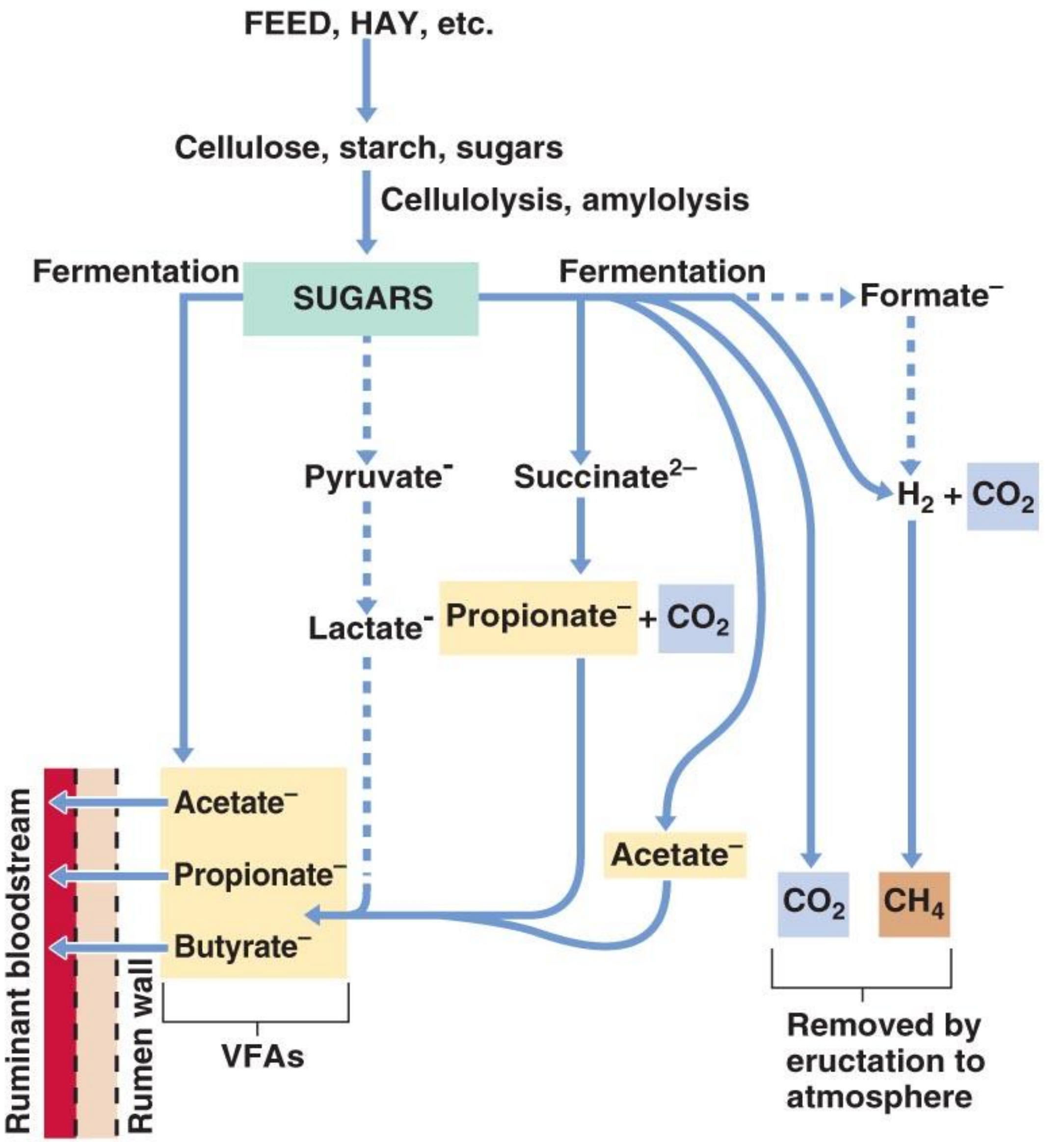
Figure 24.27a

- The rumen contains 10^{10}-10^{11}microbes/g of rumen constituents

- Microbial fermentation in the rumen is mediated by celluloytic microbes that hydrolyze cellulose to free glucose that is then fermented, producing volatile fatty acids (e.g., acetic, propionic, butyric) and the CH_4 and CO_2

- Fatty acids pass through rumen wall into bloodstream and are utilized by the animal as its main energy source

Biochemical Reactions in the Rumen



FEED, HAY, etc.

Cellulose, starch, sugars

Cellulolysis, amylolysis

Fermentation

SUGARS

Fermentation

$Formate^-$

$Pyruvate^-$

$Succinate^{2-}$

H_2 + CO_2

$Lactate^-$

$Propionate^-$ + CO_2

$Acetate^-$

$Propionate^-$

$Butyrate^-$

$Acetate^-$

CO_2

CH_4

Ruminant bloodstream

Rumen wall

VFAs

Removed by eructation to atmosphere

Overall stoichiometry of rumen fermentation:

57.5 glucose ⟶ 65 $acetate^-$ + 20 $propionate^-$ + 15 $butyrate^-$ +

60 CO_2 + 35 CH_4 + 25 H_2O

Figure 24.28

- Rumen microbes also synthesize amino acids and vitamins for their animal host
- Rumen microbes themselves can serve as a source of protein to their host when they are directly digested
- Anaerobic bacteria dominate in the rumen

Characteristics of Some Rumen Prokaryotes

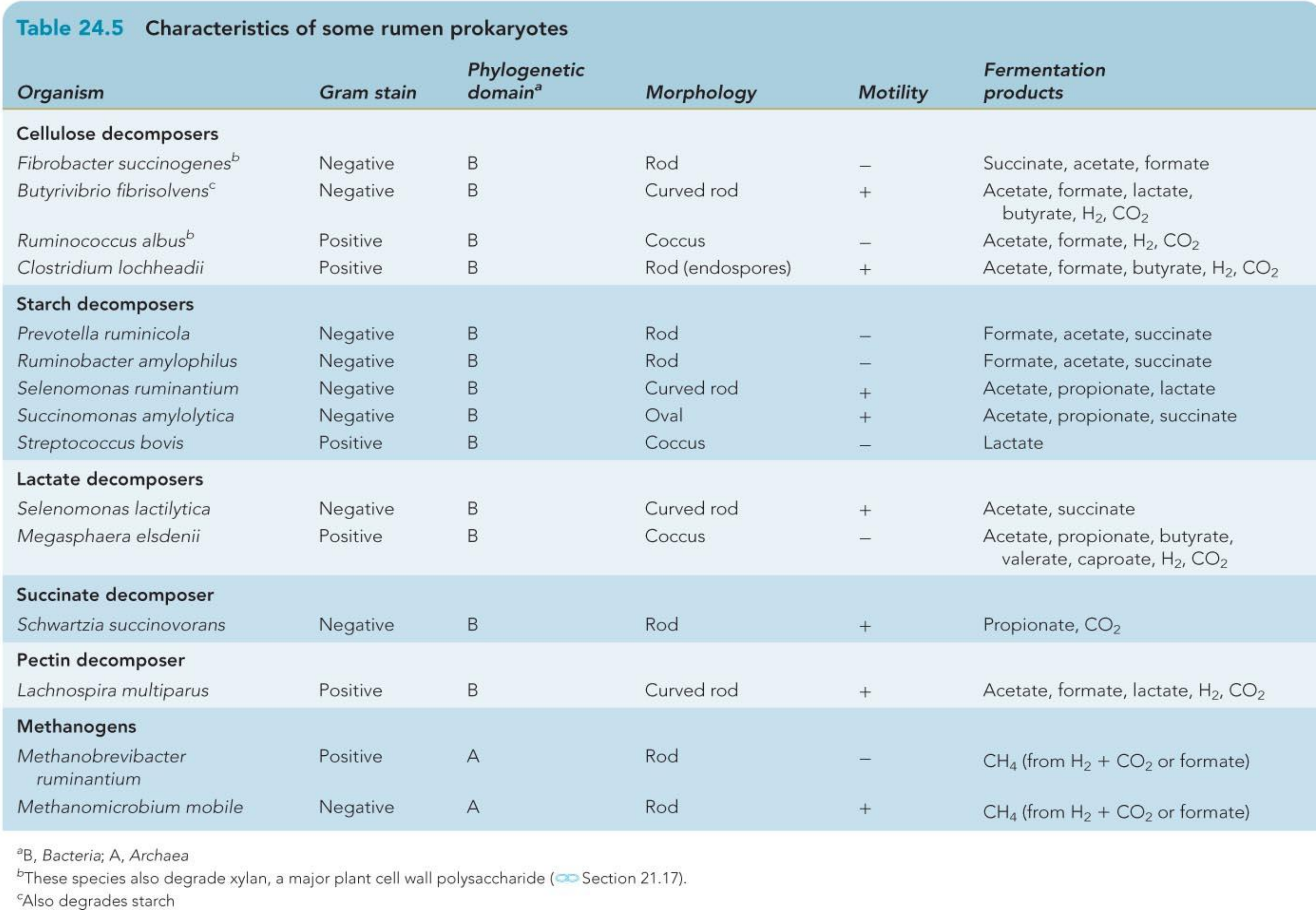


Table 24.5 Characteristics of some rumen prokaryotes

Organism	Gram stain	Phylogenetic domain[a]	Morphology	Motility	Fermentation products
Cellulose decomposers					
Fibrobacter succinogenes[b]	Negative	B	Rod	–	Succinate, acetate, formate
Butyrivibrio fibrisolvens[c]	Negative	B	Curved rod	+	Acetate, formate, lactate, butyrate, H_2, CO_2
Ruminococcus albus[b]	Positive	B	Coccus	–	Acetate, formate, H_2, CO_2
Clostridium lochheadii	Positive	B	Rod (endospores)	+	Acetate, formate, butyrate, H_2, CO_2
Starch decomposers					
Prevotella ruminicola	Negative	B	Rod	–	Formate, acetate, succinate
Ruminobacter amylophilus	Negative	B	Rod	–	Formate, acetate, succinate
Selenomonas ruminantium	Negative	B	Curved rod	+	Acetate, propionate, lactate
Succinomonas amylolytica	Negative	B	Oval	+	Acetate, propionate, succinate
Streptococcus bovis	Positive	B	Coccus	–	Lactate
Lactate decomposers					
Selenomonas lactilytica	Negative	B	Curved rod	+	Acetate, succinate
Megasphaera elsdenii	Positive	B	Coccus	–	Acetate, propionate, butyrate, valerate, caproate, H_2, CO_2
Succinate decomposer					
Schwartzia succinovorans	Negative	B	Rod	+	Propionate, CO_2
Pectin decomposer					
Lachnospira multiparus	Positive	B	Curved rod	+	Acetate, formate, lactate, H_2, CO_2
Methanogens					
Methanobrevibacter ruminantium	Positive	A	Rod	–	CH_4 (from $H_2 + CO_2$ or formate)
Methanomicrobium mobile	Negative	A	Rod	+	CH_4 (from $H_2 + CO_2$ or formate)

[a] B, *Bacteria*; A, *Archaea*

[b] These species also degrade xylan, a major plant cell wall polysaccharide (Section 21.17).

[c] Also degrades starch

- Abrupt changes in an animal’s diet can result in changes in the rumen flora
- Rumen acidification (acidosis) is one consequence of such a change

- Anaerobic protists and fungi are also abundant in the rumen

  - Many perform similar metabolisms as their prokaryotic counterparts

Commensalism

- one organism is benefited and the other is unaffected by this type of relationship
  - many of the microorganisms that make up our normal flora
    - these bacteria live on secretions and sloughed off cells
    - they bring no benefit to the host and yet the microorganisms benefit greatly from the environment they inhabit

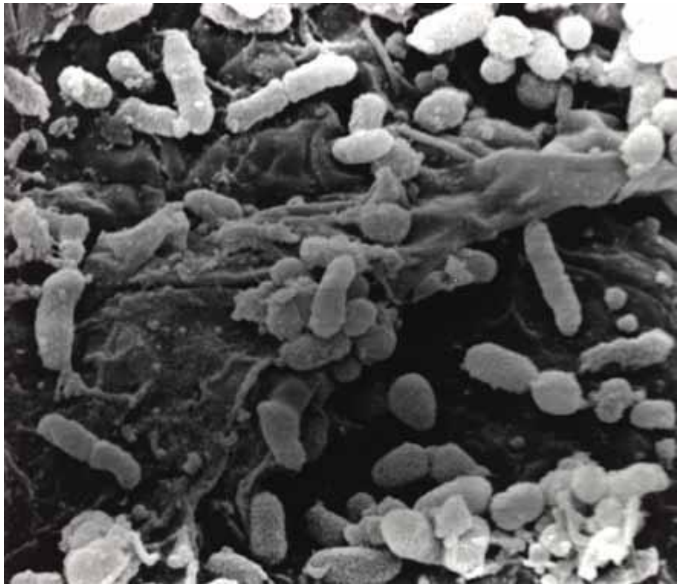
Normal Flora

- microorganisms that colonize a host without causing disease
- two types of normal flora exist
  - resident flora are microorganisms that inhabit sites on the body for extended periods
  - transient flora are microorganisms that are temporary

Normal Flora

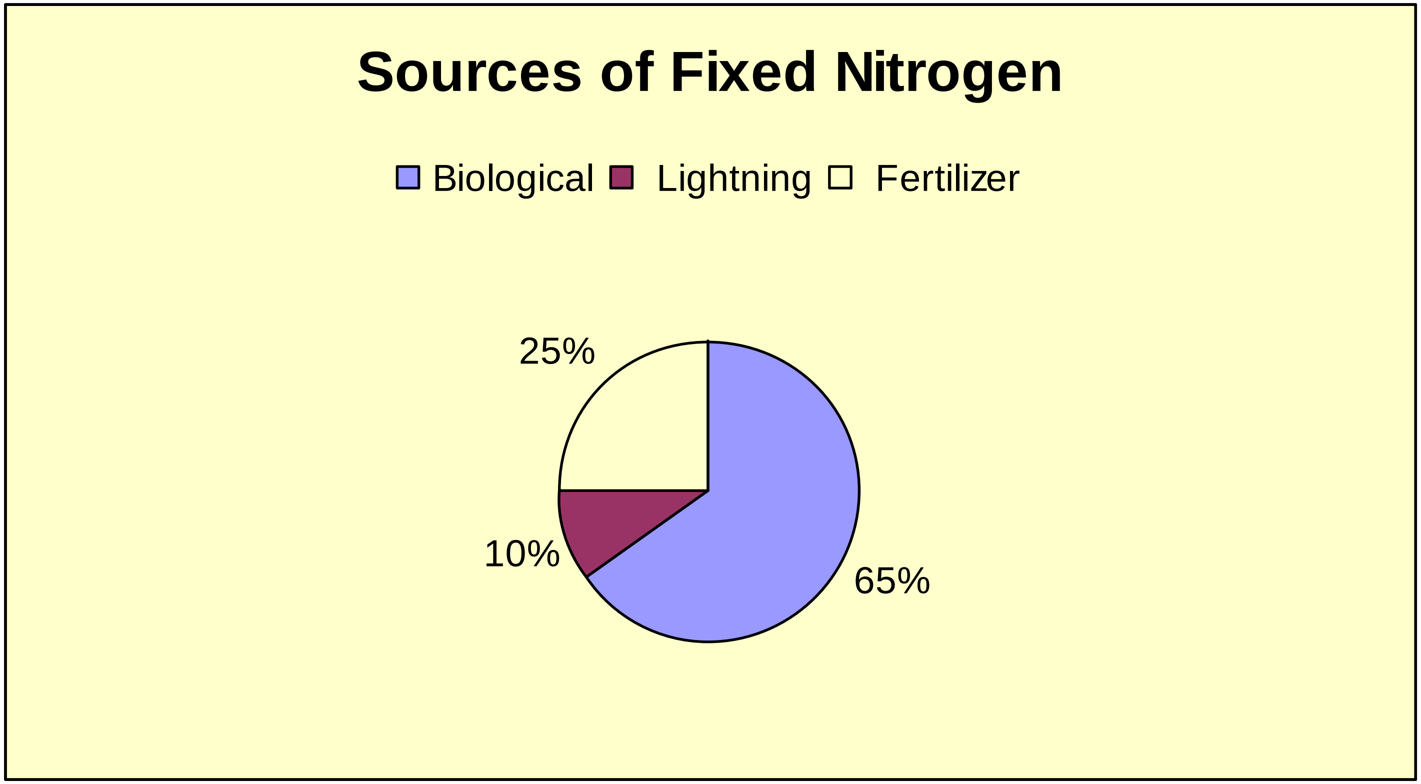
- the presence of normal flora
  - cover potential adherence sites for invading microorganisms
  - consume the available nutrients
  - produce compounds toxic to other microorganisms

bacteria found on skin



Beneficial root-microbe interactions

- Atmosphere contains 10^{15} tons N_2 gas
  - Biological nitrogen fixation
  - Minimum of 70 million tons N fixed/year

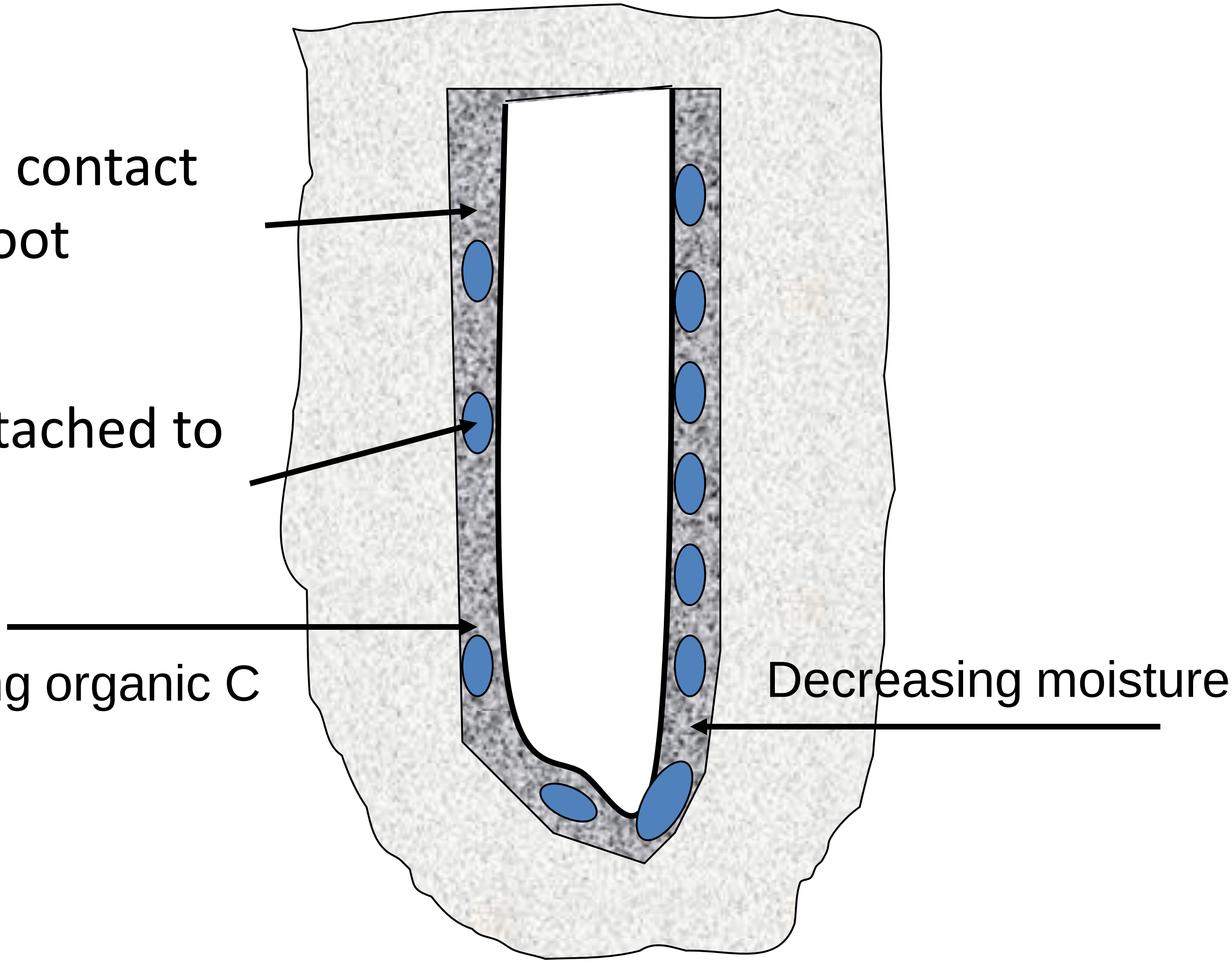


Background

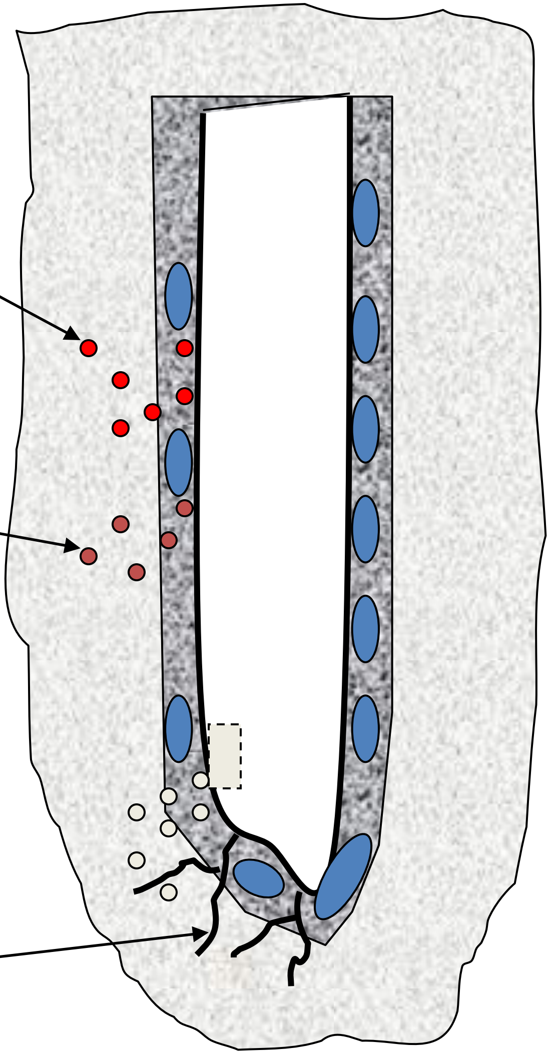
- Symbiotic Relationships
  - Both host and parasite benefit
  - Ex. Rhizobia (Symbiont) and **Legumes** (Host)
  - Rhizobia: sugars, proteins, and oxygen
  - Plant: usable nitrogen

Rhizosphere

- Rhizoplane
  - soil in direct contact with plant root
- Endophytes
  - microbes attached to root surface



Organic material in rhizosphere



- Exudates
  - low molecular weight compounds released from plant cells in a non-metabolic manner (leakage)
- Secretions
  - compounds metabolically released from plant cells
- Lysates
  - compounds released from moribund cells during autolysis
- Plant mucilage
  - plant polysacchrides

Densities of microorganisms in rhizosphere

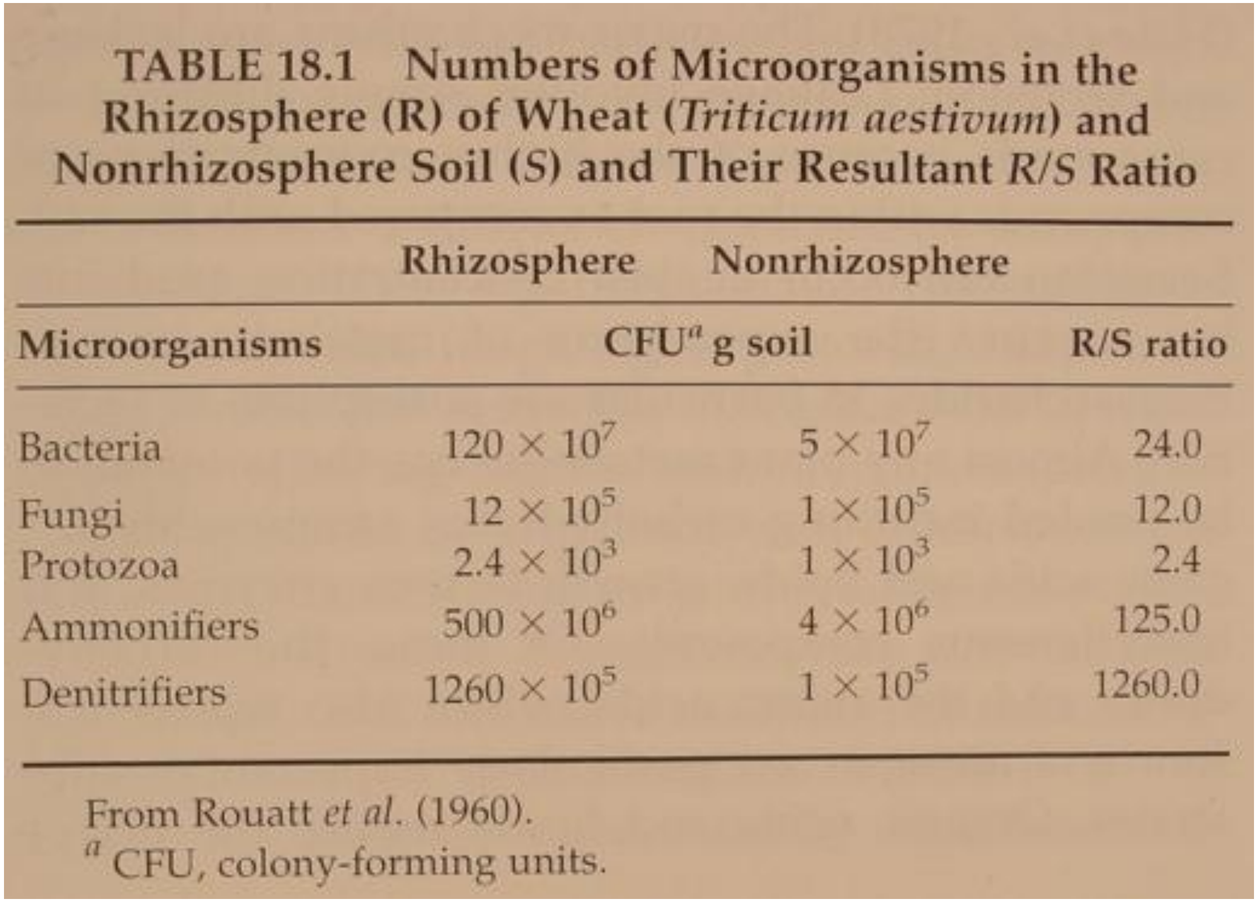
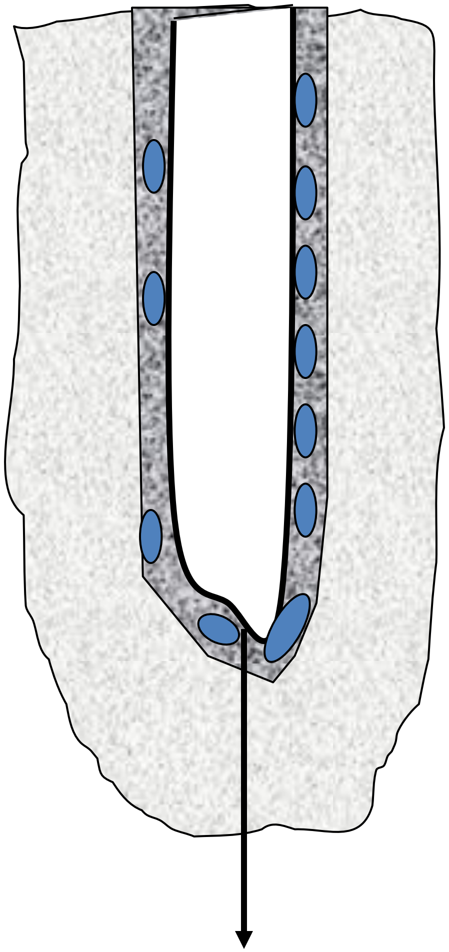


TABLE 18.1 Numbers of Microorganisms in the Rhizosphere (R) of Wheat (*Triticum aestivum*) and Nonrhizosphere Soil (*S*) and Their Resultant *R/S* Ratio

	Rhizosphere	Nonrhizosphere	
Microorganisms	CFU[a] g soil		R/S ratio
Bacteria	120 × 10^7	5 × 10^7	24.0
Fungi	12 × 10^5	1 × 10^5	12.0
Protozoa	2.4 × 10^3	1 × 10^3	2.4
Ammonifiers	500 × 10^6	4 × 10^6	125.0
Denitrifiers	1260 × 10^5	1 × 10^5	1260.0

From Rouatt *et al*. (1960).

[a] CFU, colony-forming units.



Decreasing microbial diversity

The Root Nodule Bacteriod

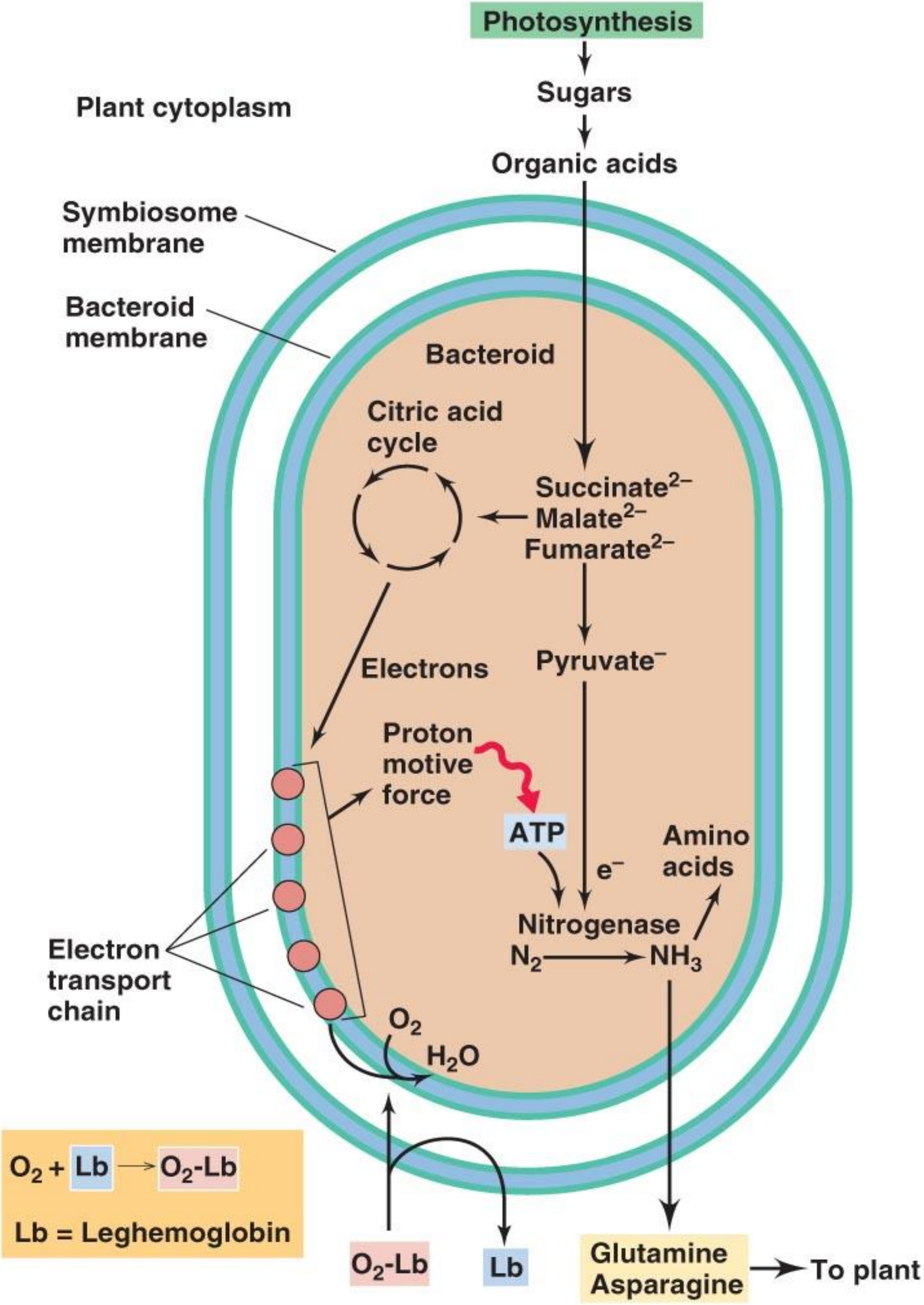


Figure 24.49

Importance of legume-rhizobia symbiosis

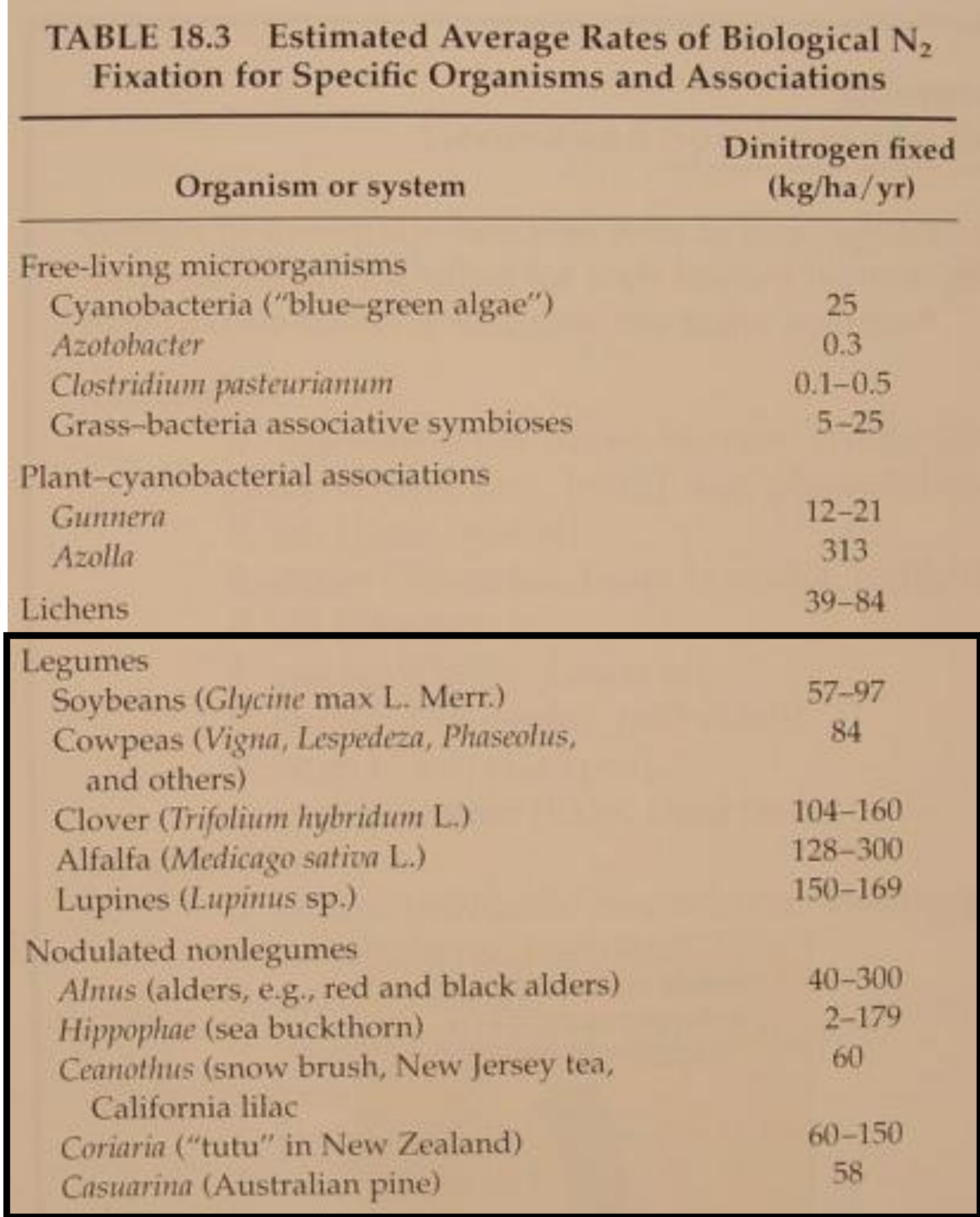
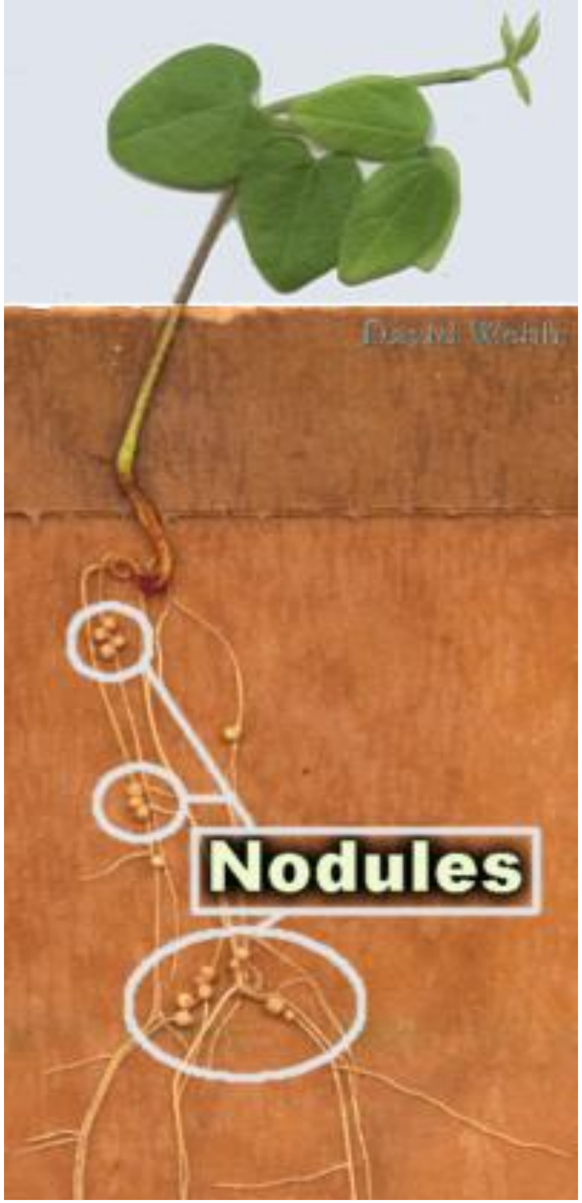
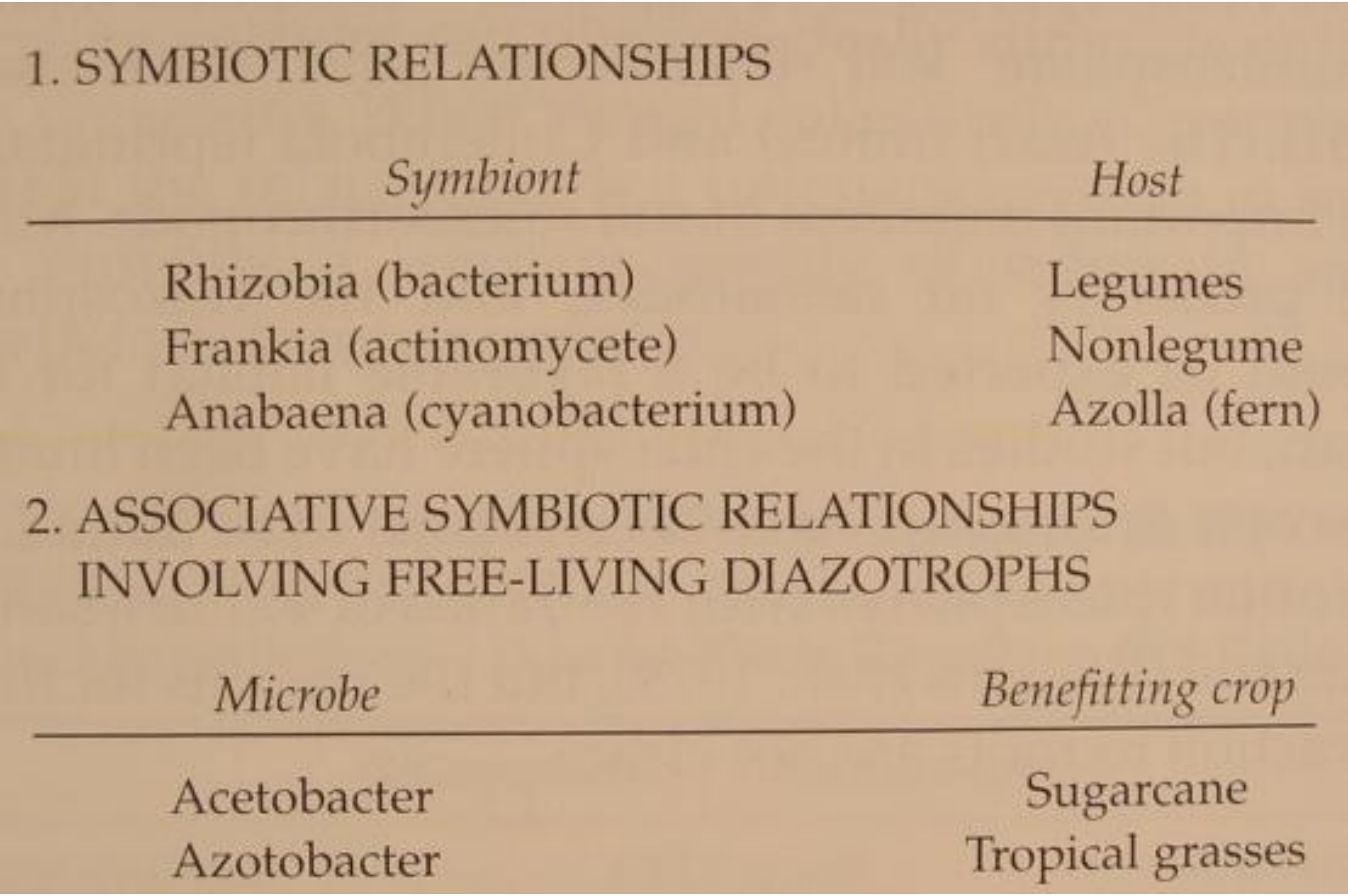


TABLE 18.3 Estimated Average Rates of Biological N_2 Fixation for Specific Organisms and Associations

Organism or system	Dinitrogen fixed (kg/ha/yr)
Free-living microorganisms	
Cyanobacteria ("blue–green algae")	25
Azotobacter	0.3
Clostridium pasteurianum	0.1–0.5
Grass–bacteria associative symbioses	5–25
Plant–cyanobacterial associations	
Gunnera	12–21
Azolla	313
Lichens	39–84
Legumes	
Soybeans (*Glycine* max L. Merr.)	57–97
Cowpeas (*Vigna, Lespedeza, Phaseolus,* and others)	84
Clover (*Trifolium hybridum* L.)	104–160
Alfalfa (*Medicago sativa* L.)	128–300
Lupines (*Lupinus* sp.)	150–169
Nodulated nonlegumes	
Alnus (alders, e.g., red and black alders)	40–300
Hippophae (sea buckthorn)	2–179
Ceanothus (snow brush, New Jersey tea, California lilac	60
Coriaria ("tutu" in New Zealand)	60–150
Casuarina (Australian pine)	58

Symbiotic N-fixation





1. SYMBIOTIC RELATIONSHIPS

Symbiont	*Host*
Rhizobia (bacterium)	Legumes
Frankia (actinomycete)	Nonlegume
Anabaena (cyanobacterium)	Azolla (fern)

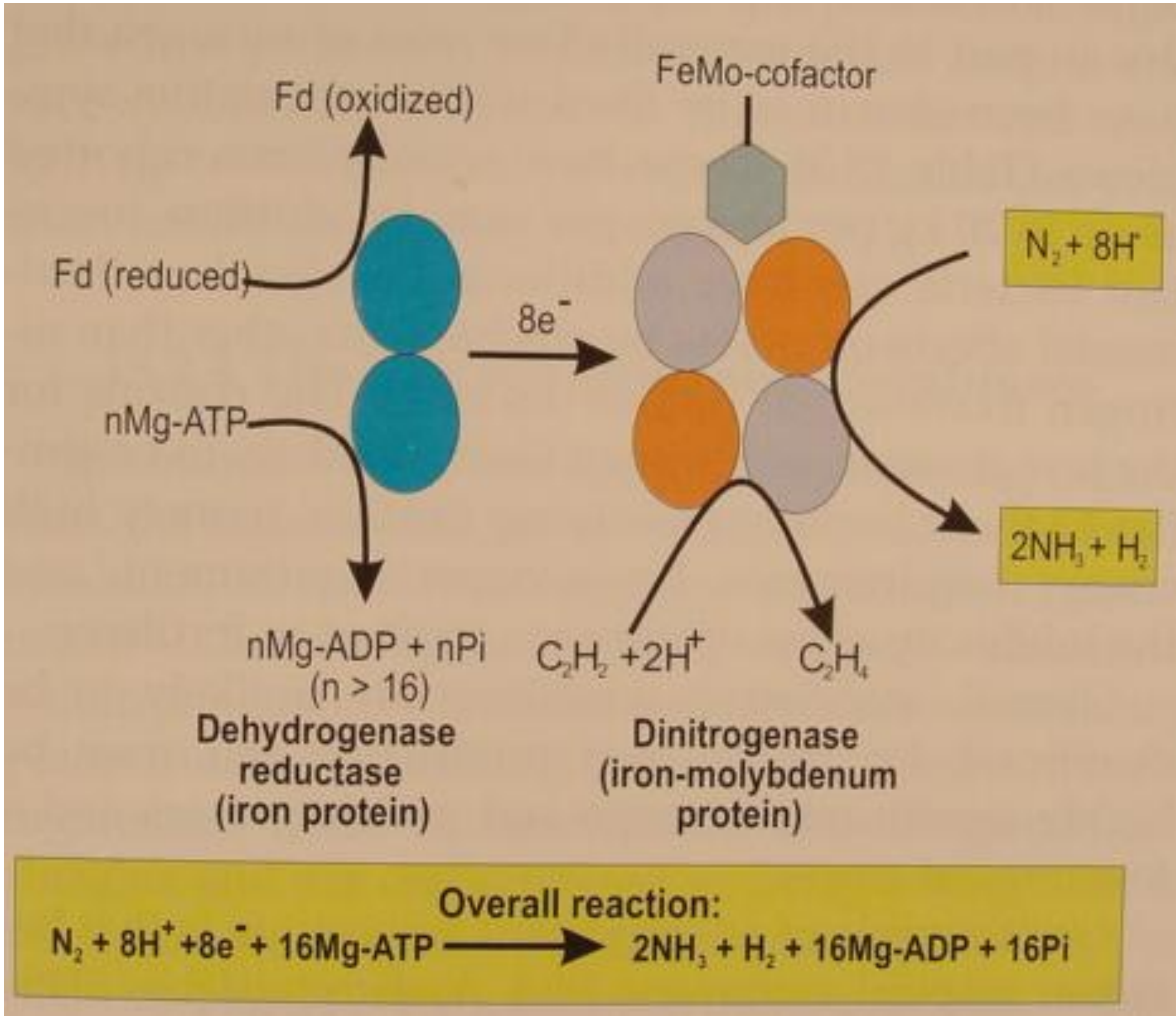
2. ASSOCIATIVE SYMBIOTIC RELATIONSHIPS INVOLVING FREE-LIVING DIAZOTROPHS

Microbe	*Benefitting crop*
Acetobacter	Sugarcane
Azotobacter	Tropical grasses

Beneficial root-microbe interactions

- Biological nitrogen fixation

$N_2 + 3H_2 \rightarrow 2NH_3$



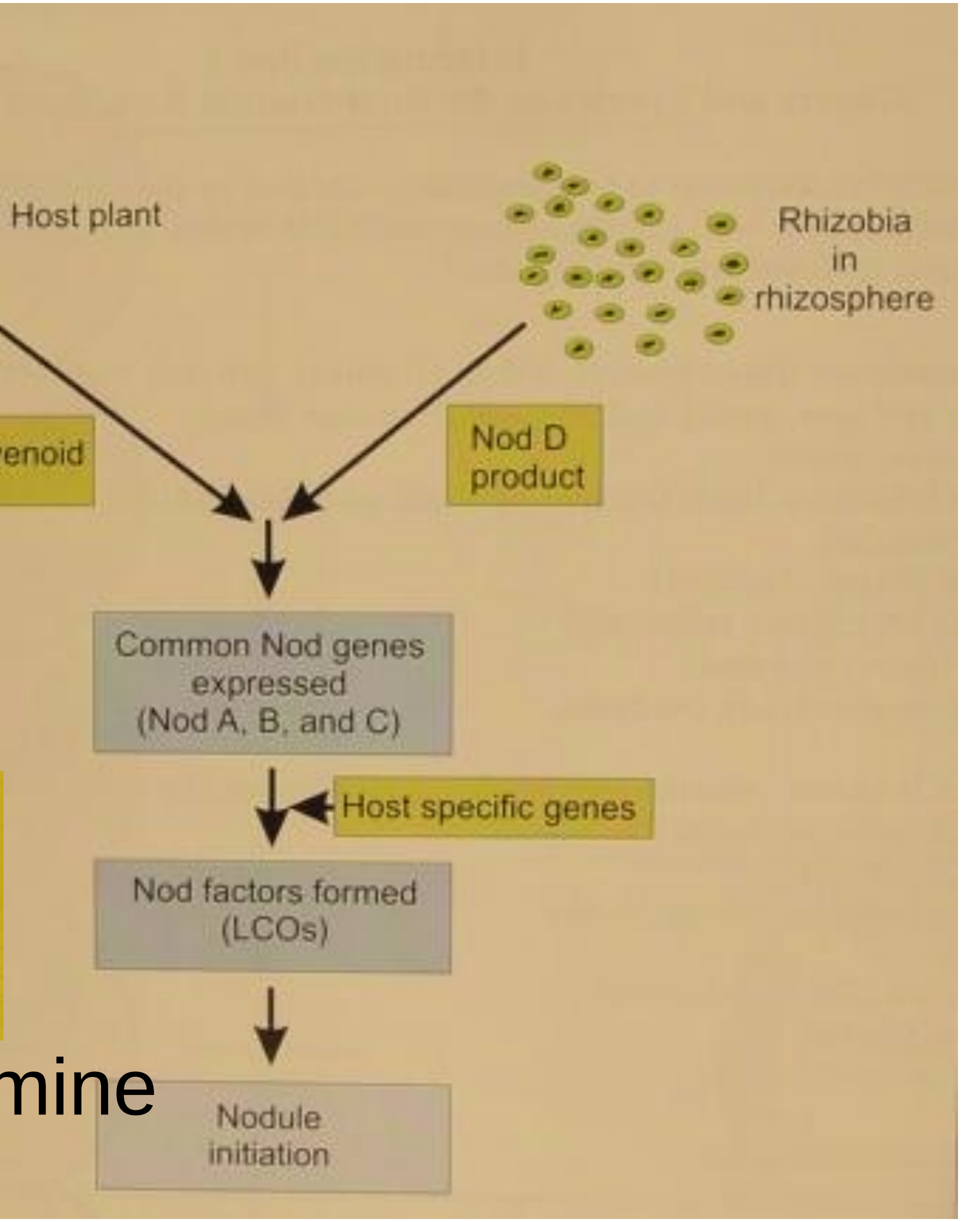
Overall reaction:

$$N_2 + 8H^+ + 8e^- + 16Mg\text{-}ATP \longrightarrow 2NH_3 + H_2 + 16Mg\text{-}ADP + 16Pi$$

Root Nodule Initiation

- Two types of Nodule
  - Effective
  - Ineffective
- Two groups of Genes Necessary
  - Common Nodulation Genes (*nodABCD*)
    - *nodD* only gene expressed in absence of a suitable host
  - Host Specific Nodulation Genes
- Legumes release flavonoids
- Triggers production of Nod Factors (Lipochitooligosaccharide) by bacteria
- Flavenoids interact with *nodD* product
- Expression of *nodABC* genes

Plant-rhizobia genetic interactions



Host plant

Rhizobia in rhizosphere

Flavenoid

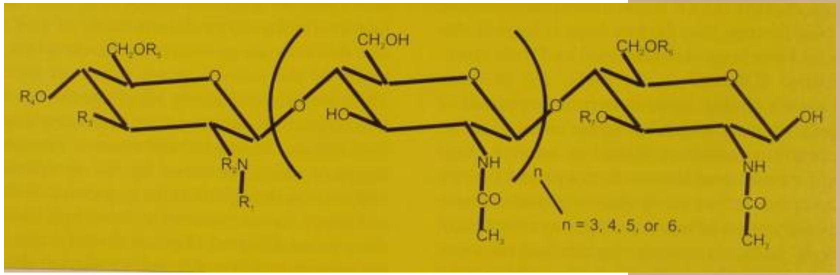
Nod D product

Common Nod genes expressed (Nod A, B, and C)

Host specific genes

Nod factors formed (LCOs)

Nodule initiation



Poly-N-acetyl-glucosamine

Root Nodule
Fixation

- Nodulins produced during maturation include:Leghemoglobin (protects nitrogenase enzyme) and also nitrogenase and gluatmine synthetase
- Fixation usually occurs after about 15 days
- Indeterminate
  - Fixation: ammonia Exported: asparagine
- Determinate
  - Fixation: ammonia Exported: purine

Enhancing the Symbiosis

- Natural symbiosis is reasonably effective
  - Free-living nitrogen fixation gives 25kg/hectare/year
  - Symbiotic nitrogen fixation gives 100kg/hectare/crop
- Current enhancements
  - Application of rhizobial inoculants
    - Peat-based carrier with 10^9 rhizobia/gram of peat
      - Application directly to seeds, then planting
    - Application to seed furrow
    - Crop rotation/breeding

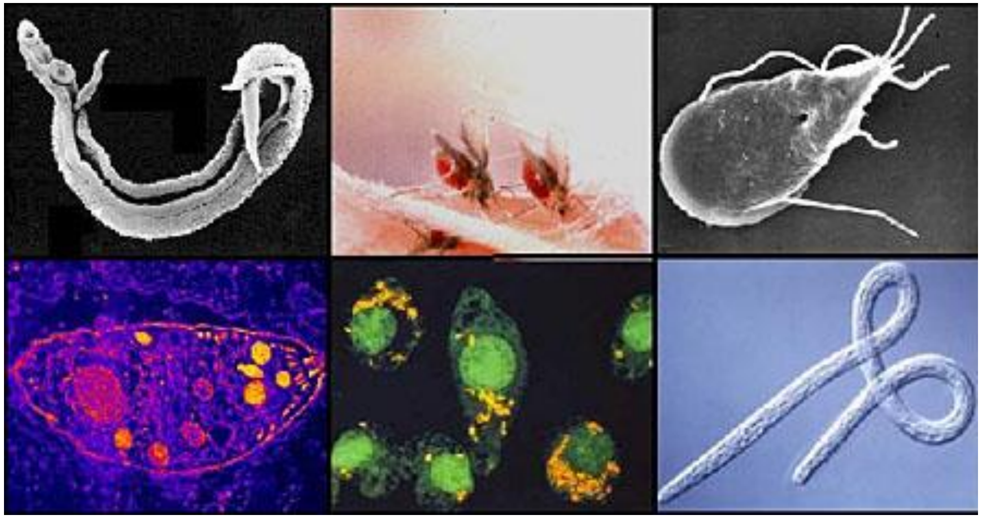
Implications

Crop Production

Land Remediation

Parasitism

- one organism benefits at the expense of the other
  - – all pathogens are parasites



parasitic microorganisms

Principles of Infectious Disease

- a parasitic relationship between a microorganism and a host is called an infection
  - infections can be subclinical or inapparent: meaning no symptoms or the symptoms are so mild as to be noticed
- infection that causes impairment of body function is called disease

Principles of Infectious Disease

- **pathogenicity** is the ability of a microorganism to cause disease by overcoming the host defenses
    - a **primary pathogen**: microorganism capable of causing disease in a healthy host
    - **opportunistic pathogen**: *usually* nonpathogenic,

  but that can become pathogenic under certain circumstances)

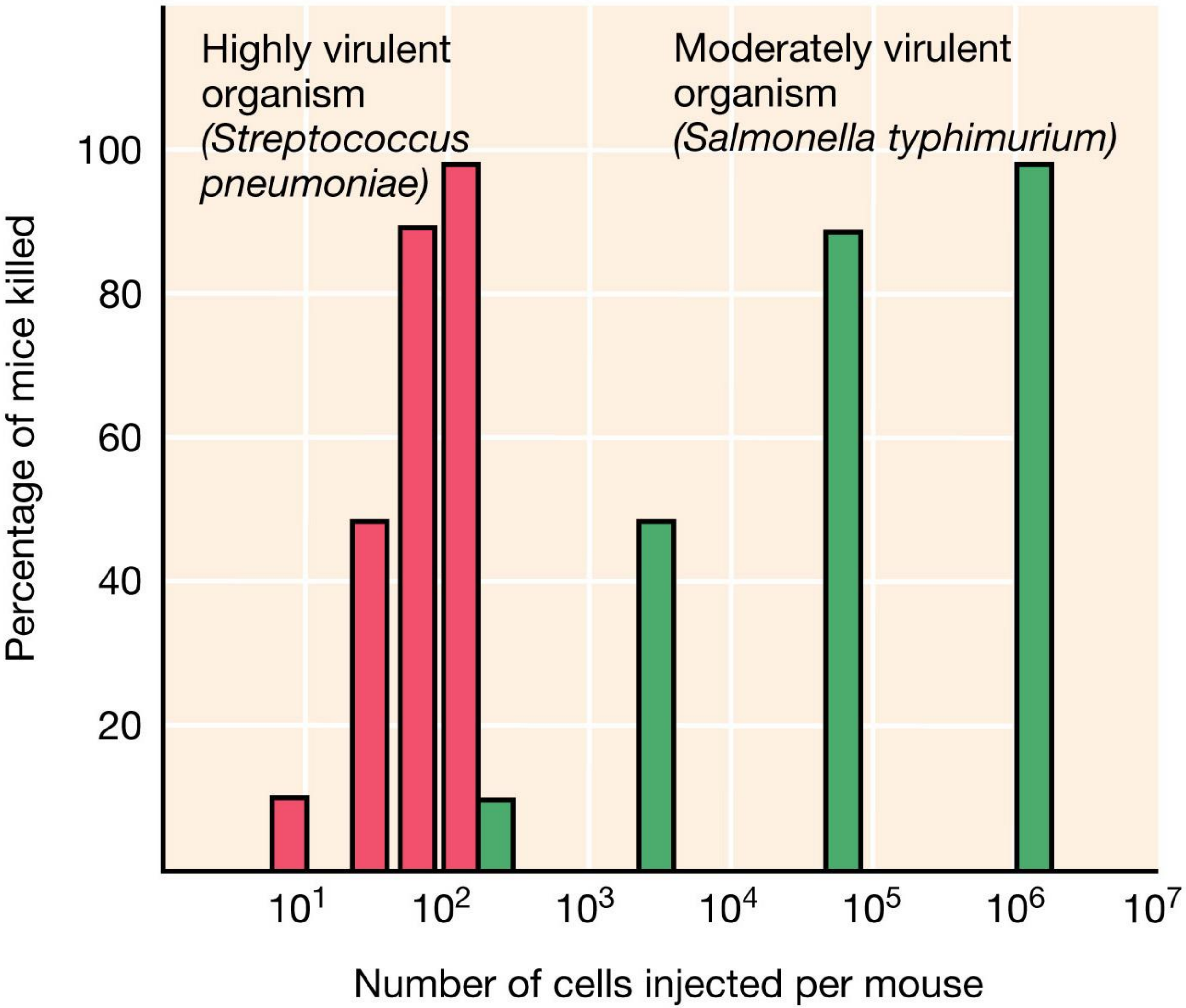
  is only capable of causing disease when the immune system is overcome, or the organism is introduced to an unusual location

        - opportunistic organisms can be part of the normal flora or found in the environment

Principles of Infectious Disease

- the more *virulent* a pathogen is the more disease promoting attributes it possesses
  - virulence factors are substances or features of a microorganism that help it infect and cause disease
  - they may include
    - ability to adhere
    - ability to overcome host defense
    - ability to evade host defense

Virulence- the degree of pathology caused by the organism



Infectious Dose- The number of organisms needed to make a person ill or a carrier

Adherence

- to establish disease the causative agent needs to
  - adhere
    - difficult to overcome our first-line defenses so adherence is imperative
    - many bacteria have adhesions, generally found on the pili

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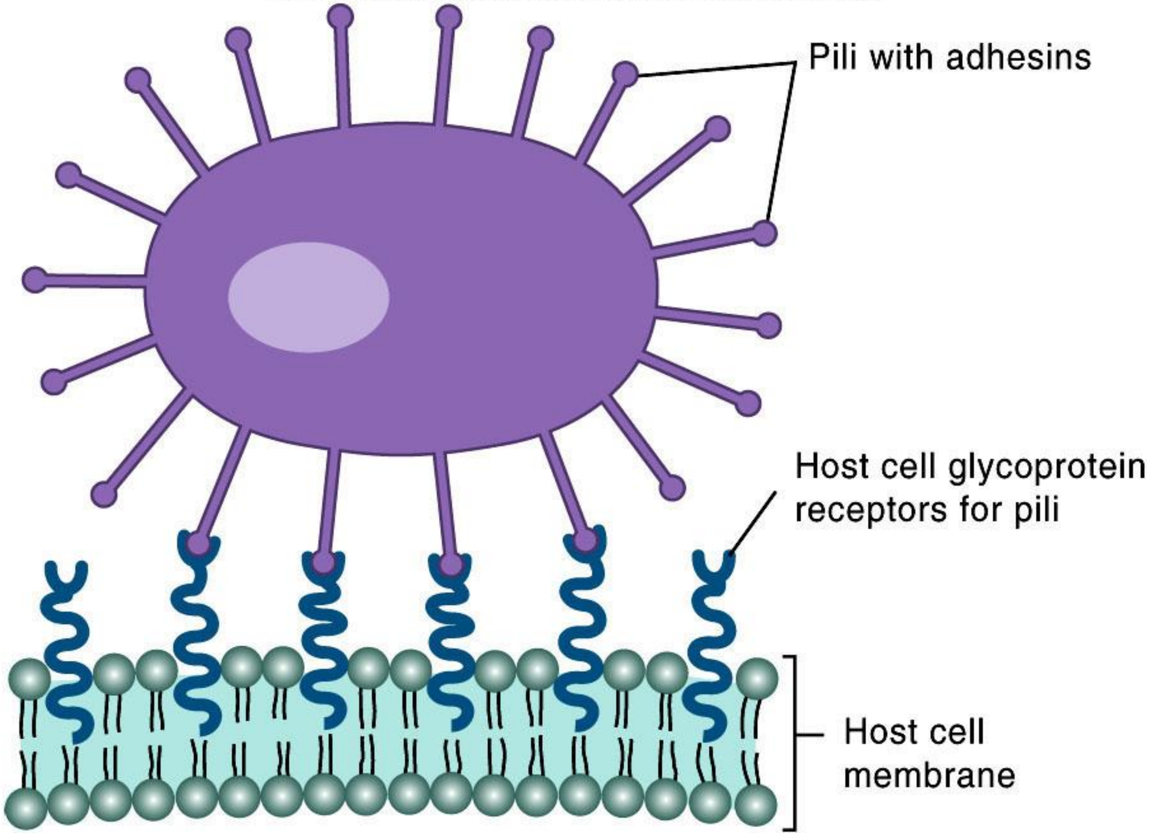


Colonization

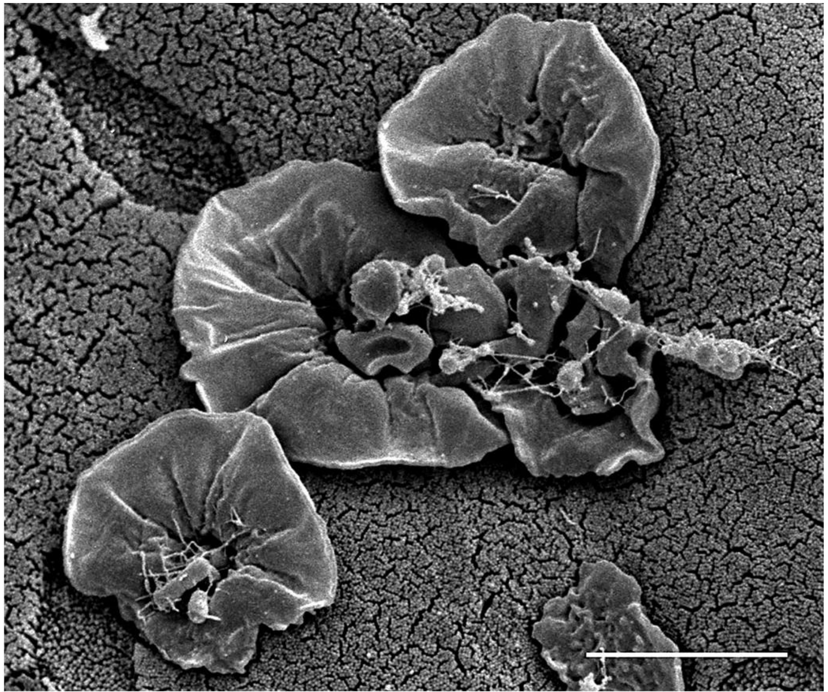
- causative agent needs to
    - multiply in order to colonize
        - to multiply, they must compete successfully with the normal flora for space and nutrients
            - toxins that may be produced by the normal flora must be overcome

Avoiding Innate System

- while some bacteria are able to cause disease while remaining on the surface of the skin or mucosa, many need to penetrate that barrier
- once this is done, those pathogens have it on easy street, exclusive rights to rich nutrition and multiplying without any competition
- penetrating the skin is extremely difficult
  - bacteria take advantage of trauma to provide a break in the skin
  - West Nile Virus is transmitted to the host through a mosquito bite, a penetration of the skin

Avoiding Innate System

- mucous membranes penetration is the most common entry for most microorganisms
  - one method that is used is referred to as *ruffling (взъерошивание, сморщивание)*
    - once the microorganism attaches to the membrane, it can direct that cell to engulf (поглощать) the bacterium: this is referred to as ruffling



ruffling on the surface of mucous membrane

Avoiding Innate and Adaptive System

- several mechanisms can be used by microorganisms to avoid the potentially lethal effects of our immune system
  - hide inside a host cell
  - interfere with the activation of complement (which attracts phagocytes)
- avoid destruction by phagocytes by simply preventing encounters with phagocytes
  - C5a peptides are an enzyme that is made by some bacteria
  - C5a peptide **destroys the complement component**
    - if the complement is not activated, neither are the phagocytes
  - some bacteria produce membrane-damaging toxins that **kill phagocytes**

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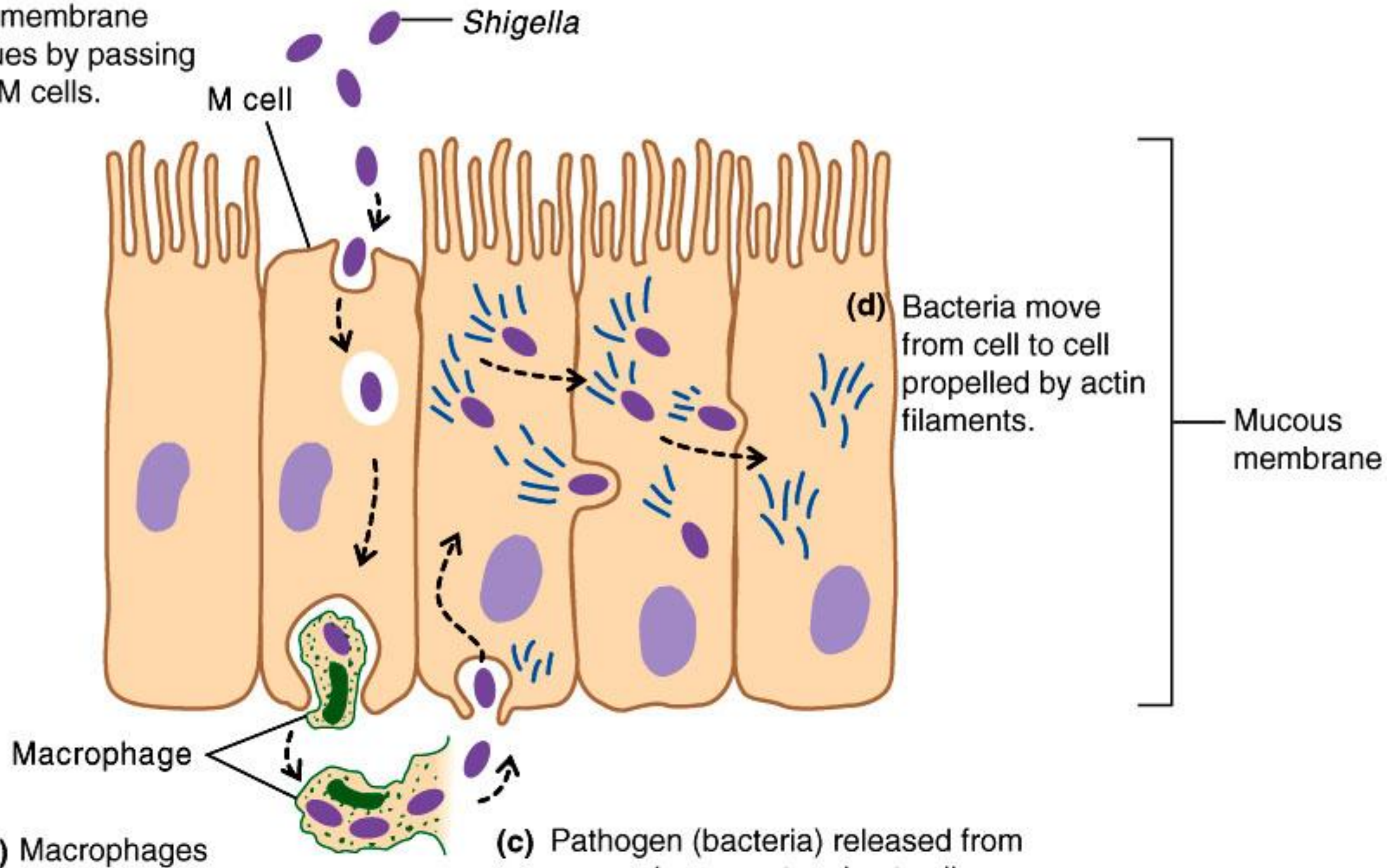

(a) *Shigella* or other bacteria cross the mucous membrane into tissues by passing through M cells.

(b) Macrophages engulf bacteria.

(c) Pathogen (bacteria) released from macrophages enters host cells by endocytosis.

(d) Bacteria move from cell to cell propelled by actin filaments.

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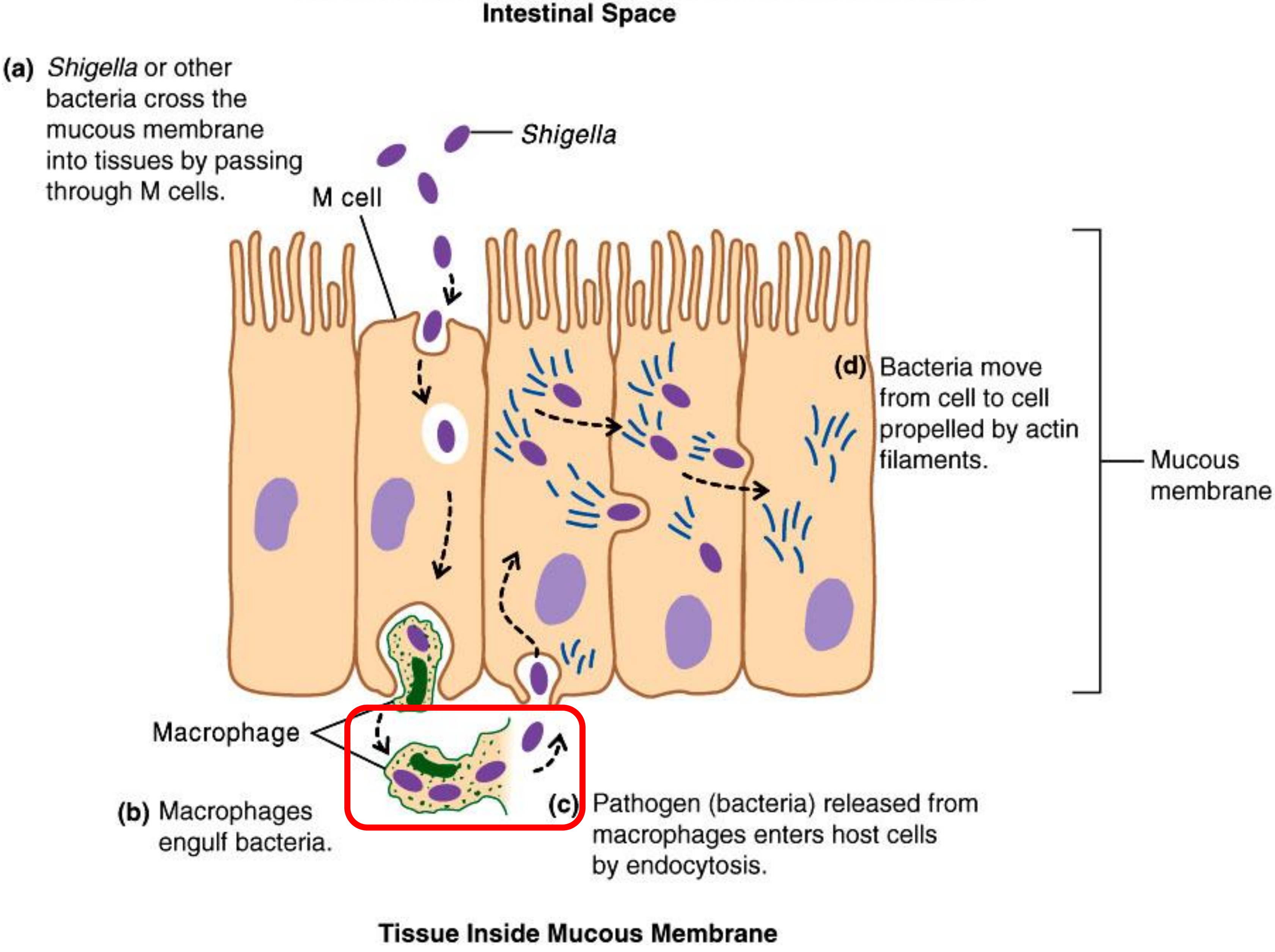

(a) *Shigella* or other bacteria cross the mucous membrane into tissues by passing through M cells.

(b) Macrophages engulf bacteria.

(c) Pathogen (bacteria) released from macrophages enters host cells by endocytosis.

(d) Bacteria move from cell to cell propelled by actin filaments.

Avoiding Innate and Adaptive System

- avoid recognition and attachment to phagocytes by
  - producing capsules to prevent phagocytosis
    - *Streprococcus pneumoniae* produces capsules

- survive in the phagocyte
  - they don't worry about being engulfed, simply enjoy the "free ride"
    - some microorganisms can escape from the phagosome before being fused with the enzyme lysosome
  - some microorganisms can block the fusion of the phagosome and lysosome
  - a few organisms can actually survive the lysosome environment

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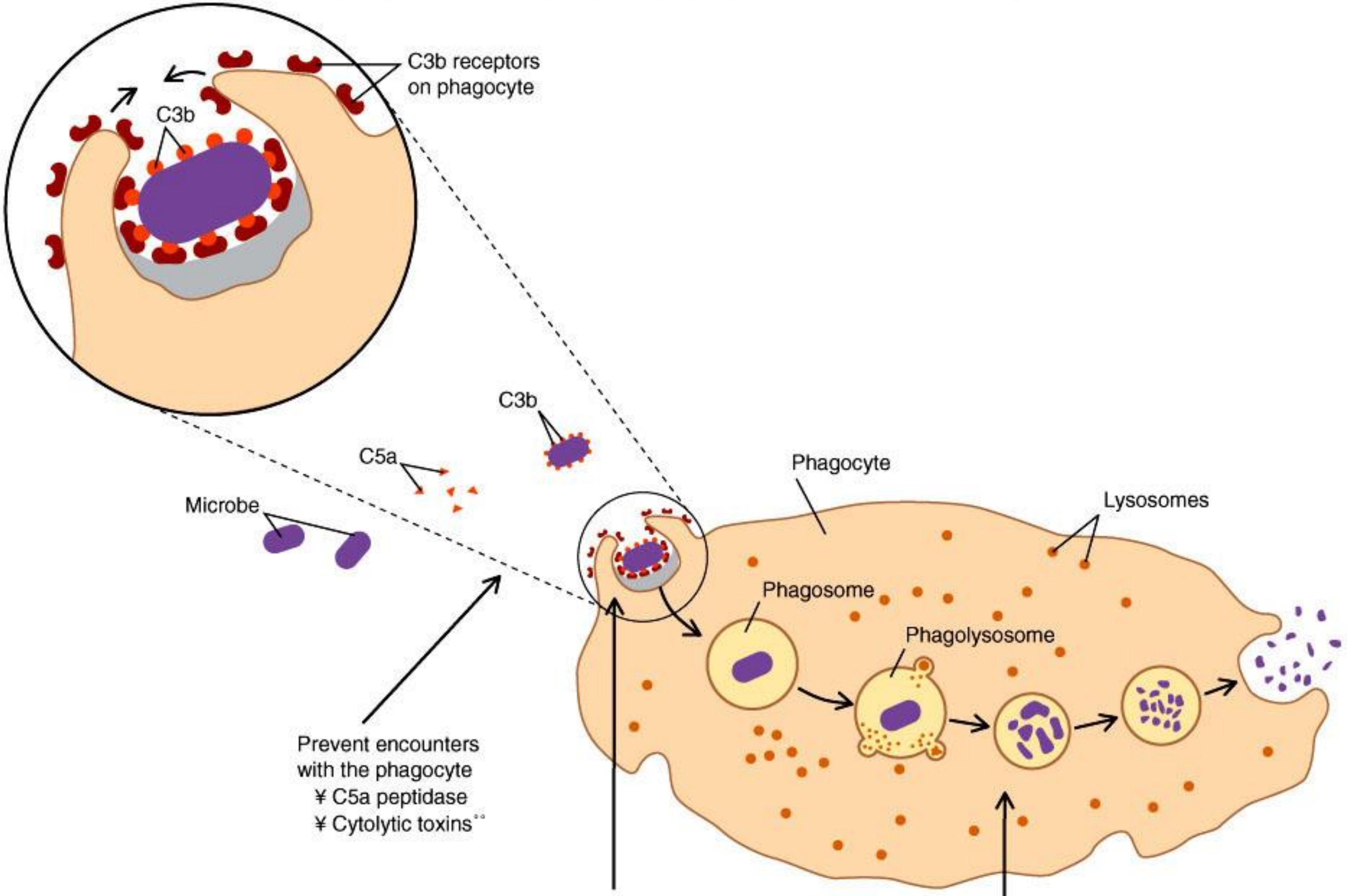


Prevent encounters with the phagocyte
- ¥ C5a peptidase
- ¥ Cytolytic toxins

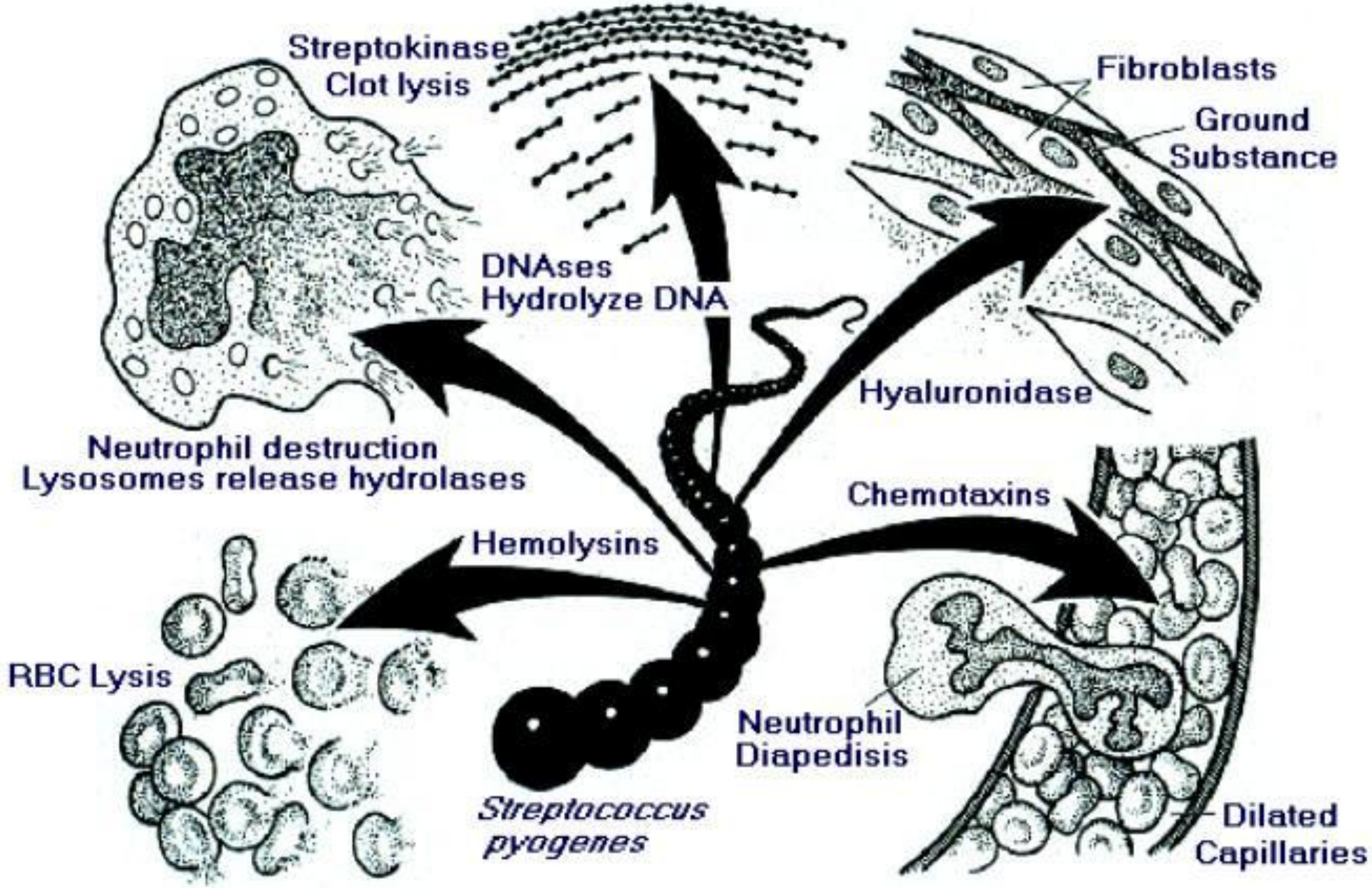
Avoid recognition and attachment
- ¥ Capsules
- ¥ M protein
- ¥ Fc receptors

Survive within the phagocyte
- ¥ Escape from the phagosome
- ¥ Prevent phagosome-lysosome fusion
- ¥ Survive within the phagosome

Avoiding the Adaptive System

- avoiding antibodies which integral to the adaptive system; this can be accomplished several ways including
  - IgA protease
    - cleaves IgA class of antibodies found in mucus and other secretions
  - antigenic variation
    - alter structure of antigens
    - stay ahead of antibody production and destruction by altering the structure the antibodies are searching for
  - mimic host molecules
    - some microorganisms have the ability to cover themselves with molecules similar to “self”

Streptococcal Virulence Factors



Indiana State University

What makes a pathogen?

Virulence Factors

Factors that improve attachment

Pili, capsules, fimbriea

Factors that allow penetration

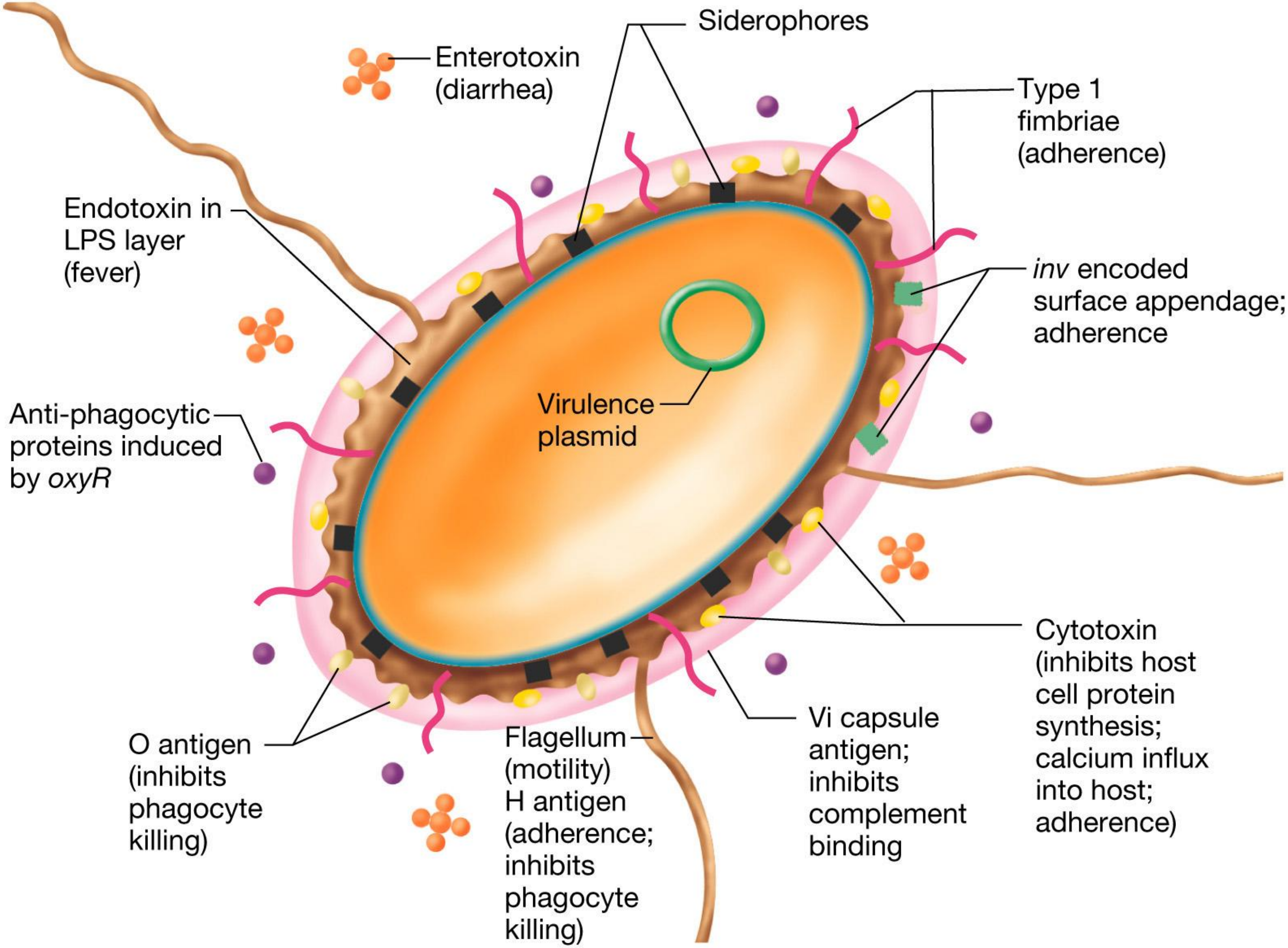
Enzymes- hyaluronidase, proteases

Factors that improve colonization

Siderophores

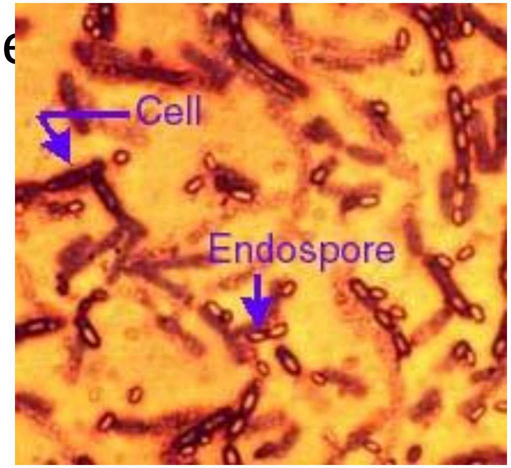
Toxins- damage host

Virulence factors important in Salmonella pathogenesis


Siderophores
Enterotoxin (diarrhea)
Type 1 fimbriae (adherence)
Endotoxin in LPS layer (fever)
inv encoded surface appendage; adherence
Virulence plasmid
Anti-phagocytic proteins induced by *oxyR*
Cytotoxin (inhibits host cell protein synthesis; calcium influx into host; adherence)
O antigen (inhibits phagocyte killing)
Flagellum (motility) H antigen (adherence; inhibits phagocyte killing)
Vi capsule antigen; inhibits complement binding

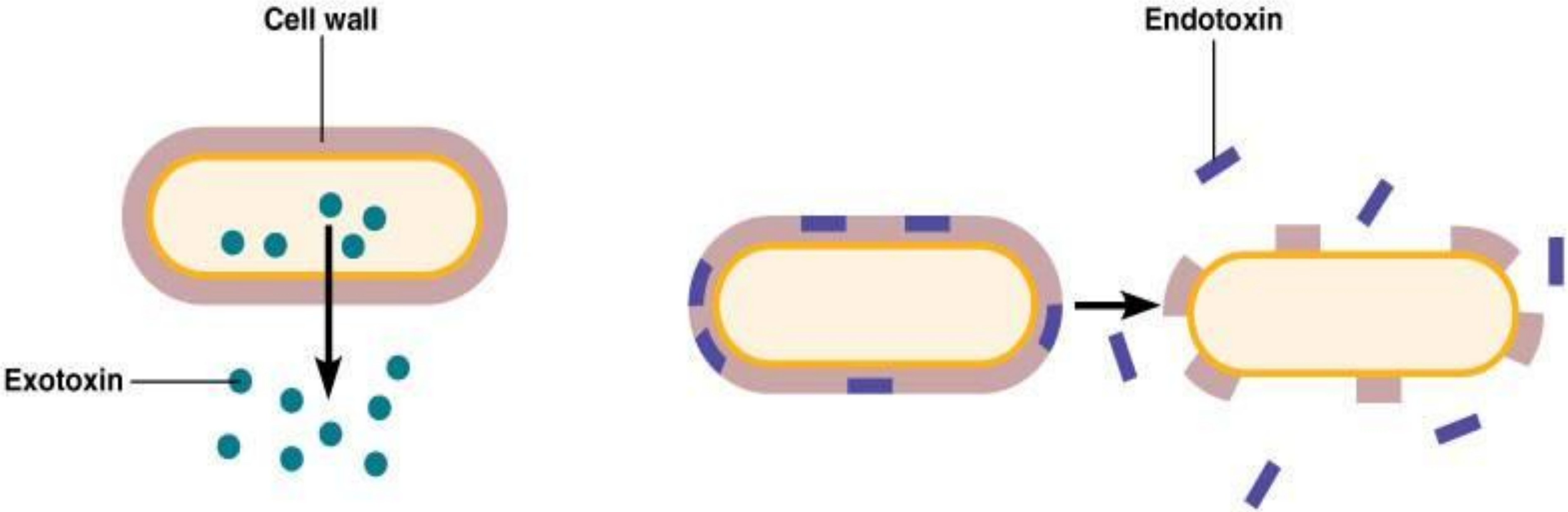
Direct Host Damage

- toxins produced by the invading pathogen cause direct damage to the host which results in disease
- toxins capable of causing damage include
  - exotoxins
    - a protein toxin released from a living ce
    - mostly found in Gram + cells



Bacillus anthraxis produces an exotoxin

Toxins



(a) **Exotoxins** are produced inside mostly gram-positive bacteria as part of their growth and metabolism. They are then secreted or released following lysis into the surrounding medium.

(b) **Endotoxins** are part of the outer portion of the cell wall (lipid A; see Figure 4.12c) of gram-negative bacteria. They are liberated when the bacteria die and the cell wall breaks apart.

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Exotoxoins

- exotoxins are secreted by the bacteria or released following lysis
- exotoxins are soluble in body fluids which makes them easily diffused into blood and then are rapidly transported throughout the body
- exotoxins work by destroying particular parts of the host cells or by inhibiting certain metabolic functions

Exotoxins

- exotoxins are highly specific
- most exotoxins are grouped according to the tissues they adversely impact
  - neurotoxins damage the nervous system
  - entereotoxins upset the intestinal system
  - cytotoxins afflict their damage on many different types of cells by disrupting cellular function of by lysing the cell

- exotoxins are among the most lethal substances known to man
- the danger with exotoxins is not the ingestion of the bacterium, but the ingestion of the toxin

Damage to the Host

- A-B toxins
  - Toxins consist of two parts
    - A subunit
      - Toxic or active part
    - B subunit
      - Binding part
        - Binds to specific host cell receptors
  - Structure offers novel approaches to development of vaccine and other therapies
    - Use toxin structure as binding a delivery system

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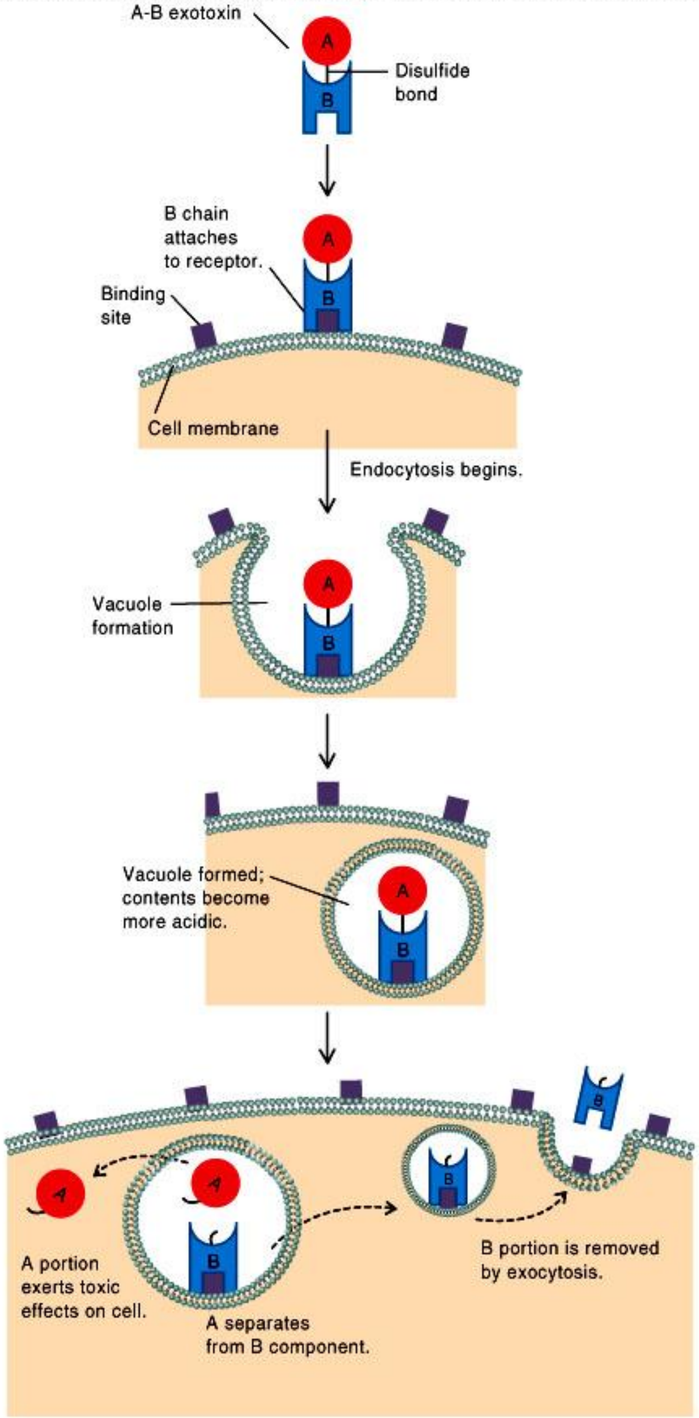
A-B exotoxin
A
B
Disulfide bond
B chain attaches to receptor.
Binding site
Cell membrane
Endocytosis begins.
Vacuole formation
Vacuole formed; contents become more acidic.
A portion exerts toxic effects on cell.
A separates from B component.
B portion is removed by exocytosis.

Damage to the Host

- Exotoxins
  - Numerous organisms produce exotoxins
  - Have very specific damaging effects
  - Among most potent toxins known
  - Often major cause of damage to infected host
  - Exotoxins are secreted by bacterium or leak into surrounding fluids following cell lysis
  - Toxins act locally or systemically
  - Made of protein
    - Makes them heat labile
  - So powerful fatal damage can occur before adequate immune response mounted
    - Passive immunity in form of antitoxin can be given as treatment

Damage to the Host

- Membrane-damaging toxins
  - Disrupt plasma membrane
    - Cause cell lysis
  - Some membrane-damaging toxins produce pores that allow fluids to enter causing cell destruction
  - Phospholipases are group of potent membrane-damaging toxins
    - Remove polar heads of phospholipid
      - Destabilizes membrane

Damage to the Host

- Superantigens
    - Override specificity of T cell response
        - Cause toxic effects due to massive release of cytokines by large number of helper T cells
    - Superantigens short-circuit normal control mechanisms of antigen process and presentation
        - Binds MHC class II and T cell receptor
            - Causes activation of 1 in 5 T cells

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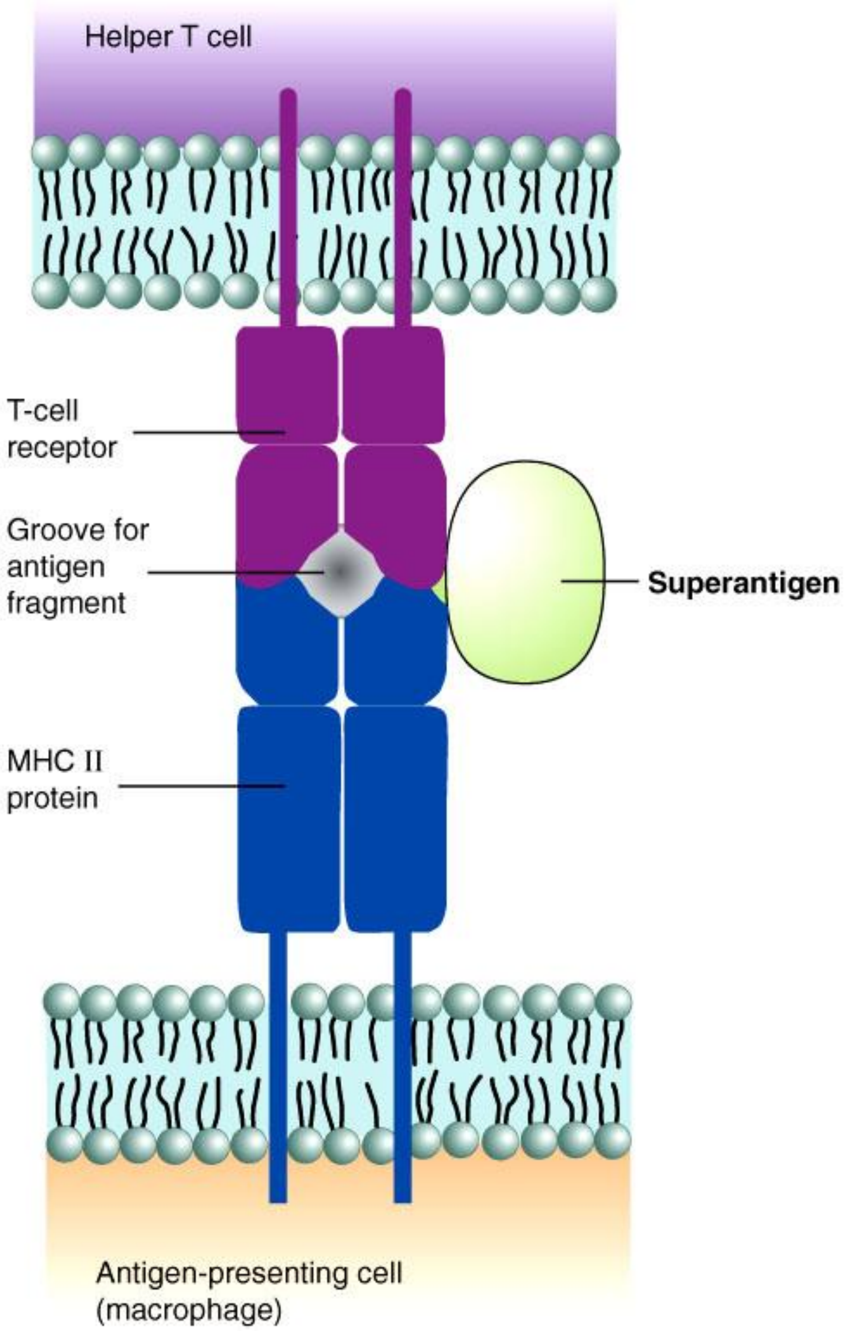

Helper T cell
T-cell receptor
Groove for antigen fragment
Superantigen
MHC II protein
Antigen-presenting cell (macrophage)

Damage to the Host

- Endotoxins
  - Endotoxins are LPS of gram-negative cell wall
    - Toxin fundamental part of gram-negative organism
  - Endotoxins *are heat stable*
  - Lipid A responsible for toxic properties
  - Symptoms associated with vigorous immune response
  - Toxin responsible for septic shock
    - a.k.a endotoxic shock

Endotoxins

- endotoxins are lipopolysaccharides (LPS) found in the lipid portion of the outer wall of Gram – bacteria
  - endotoxins are released when Gram – bacteria die and the cell wall undergoes lysis
    - antibiotics that are used to treat Gram – diseases can lyse the bacterial cells, releasing the endotoxin
      - this can lead to an immediate worsening of the symptoms
      - these symptoms usually improve as the endotoxins break down

Endotoxins

- endotoxins can also activate blood-clotting proteins, causing the formation of small blood clots
  - blood clots obstruct capillaries, resulting in decreased blood supply, which can lead to tissue death
    - this is referred to as disseminated intravascular coagulation
- endotoxins also cause fever (pyrogenic response) and rapid blood pressure decrease

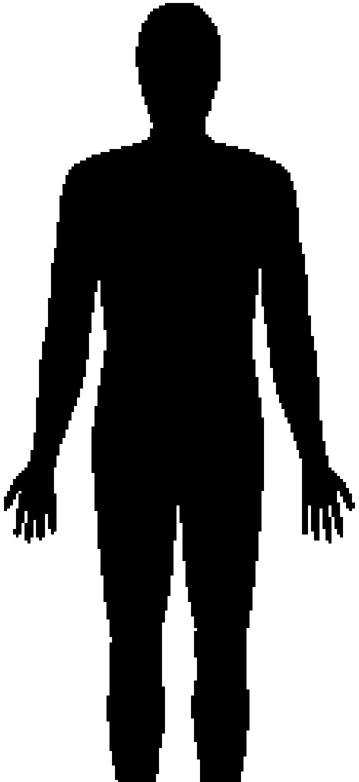
Mechanisms of Eukaryotic Pathogenesis

- these mechanisms are not clearly understood, though the mechanisms include colonization of the host, evasion of the host defenses and damage to the host
- fungi
  - these organisms are generally opportunistic, taking advantage of a weakening or change in our immune system
    - excessive growth of *Candida albicans* is often a result in immunocompromised hosts

Mechanisms of Eukaryotic Pathogenesis

- eukaryotic parasites
  - are generally found in the intestinal tract or have gained access through an insect bite
  - attach with specific receptors
  - are capable of hiding within the host cell
  - the damage they can inflict varies
    - some cause malnutrition by competing for nutrients
    - some can cause direct damage by the enzymes they produce

The Battle



Host Defenses

Physical

Chemical

Normal Flora

Nonspecific
Internal responses

Specific
Immune responses

Microbial Weapons

Virulence Factors

Capsules

Enzymes

Toxins

Siderophores

- Plants as host organisms

- A **virulent** pathogen is one that a plant has little specific defense against
- An **avirulent** pathogen is one that may harm but does not kill the host plant

Disease Development

- Infections occur through leaf scars and wounds. These give rise to small cankers in which the bacteria survive the winter.

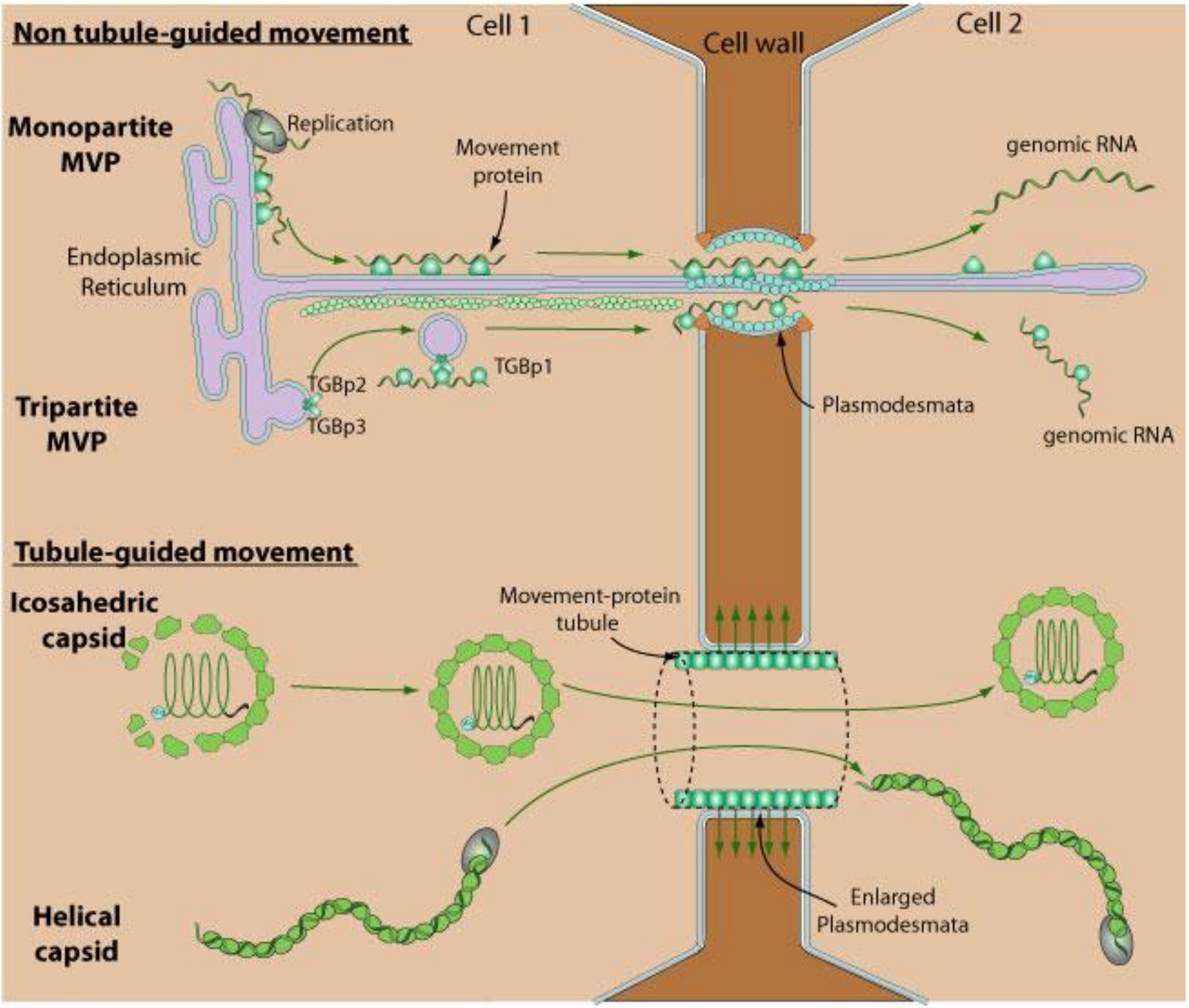
- Rain or water splash, and pruning tools spread the bacterium.

- Bacteria overwinter in active cankers, in infected buds, and on the surface of infected and healthy trees and weeds.

- The bacterium reproduces best between 21ºC and 25ºC.

- Generally disease seems to be more severe after cold winters and prolonged spring rains.

Movement of pathogens from cell to cell



Movement of pathogens from cell to cell

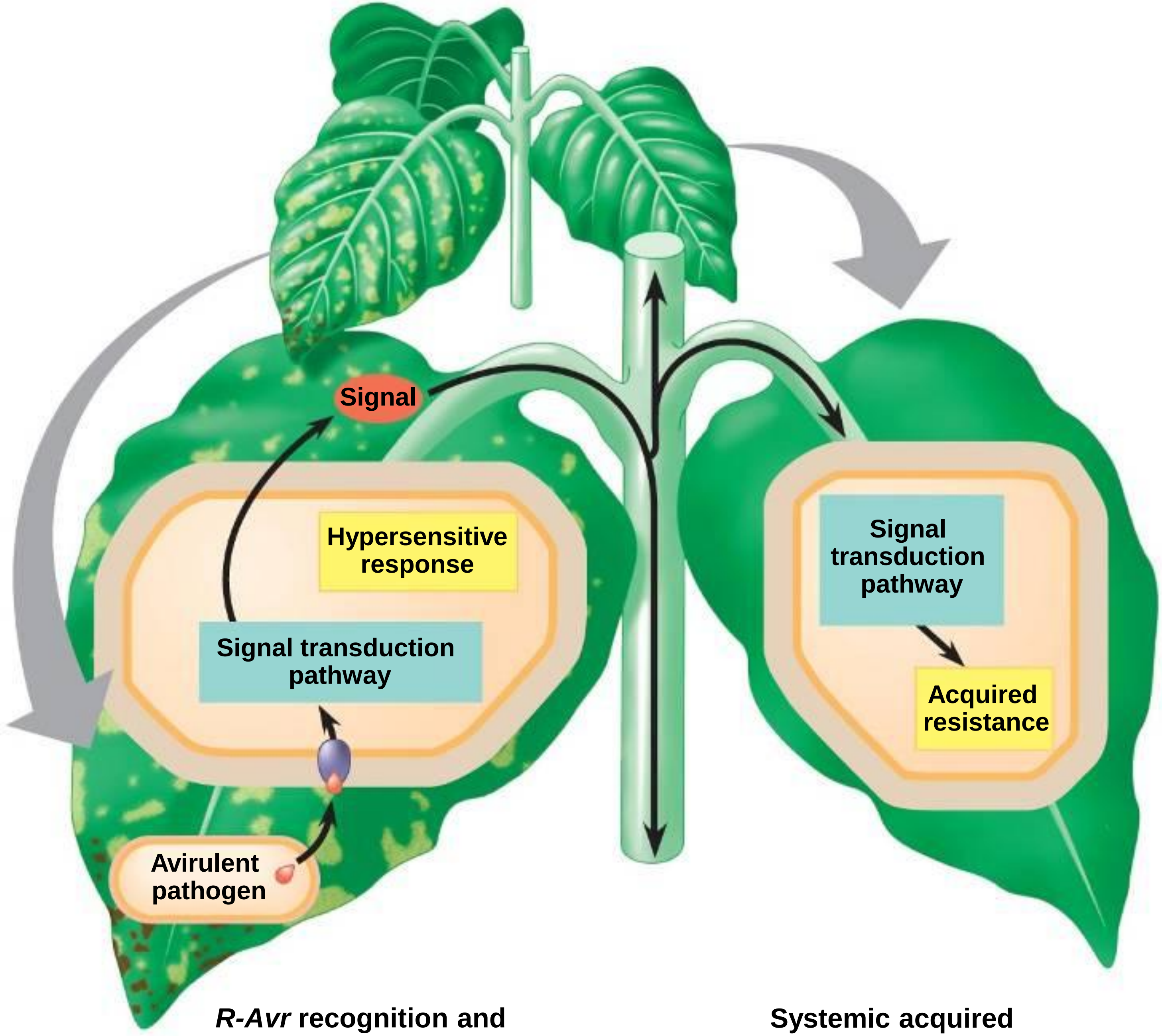
- Fungi, Bacteria, and Viruses all move through the plant in the same when following a successful penetration.

- ***Movement proteins (MP)*** are proteins dedicated to enlarging the pore size of plasmodesmata and actively transporting the pathogen into the adjacent cell.

- Thereby allowing local and systemic spread of pathogen in plants.

Defenses Against Pathogens

- A plant’s first line of defense against infection is the epidermis and periderm
- If a pathogen penetrates the dermal tissue, the second line of defense is a chemical attack that kills the pathogen and prevents its spread
- This second defense system is enhanced by the inherited ability to recognize certain pathogens



Signal

Hypersensitive response

Signal transduction pathway

Avirulent pathogen

Signal transduction pathway

Acquired resistance

R-Avr recognition and hypersensitive response

Systemic acquired resistance

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Systemic Acquired Resistance

- **Systemic acquired resistance** causes systemic expression of defense genes and is a long-lasting response
- **Salicylic acid** is synthesized around the infection site and is likely the signal that triggers systemic acquired resistance

Interaction of Ti plasmid DNA with the plant genome

Figure 17.19 An *Agrobacterium* carrying a Ti plasmid of the nopaline type induces a teratoma, in which differentiated structures develop. Photograph kindly provided by Jeff Schell.

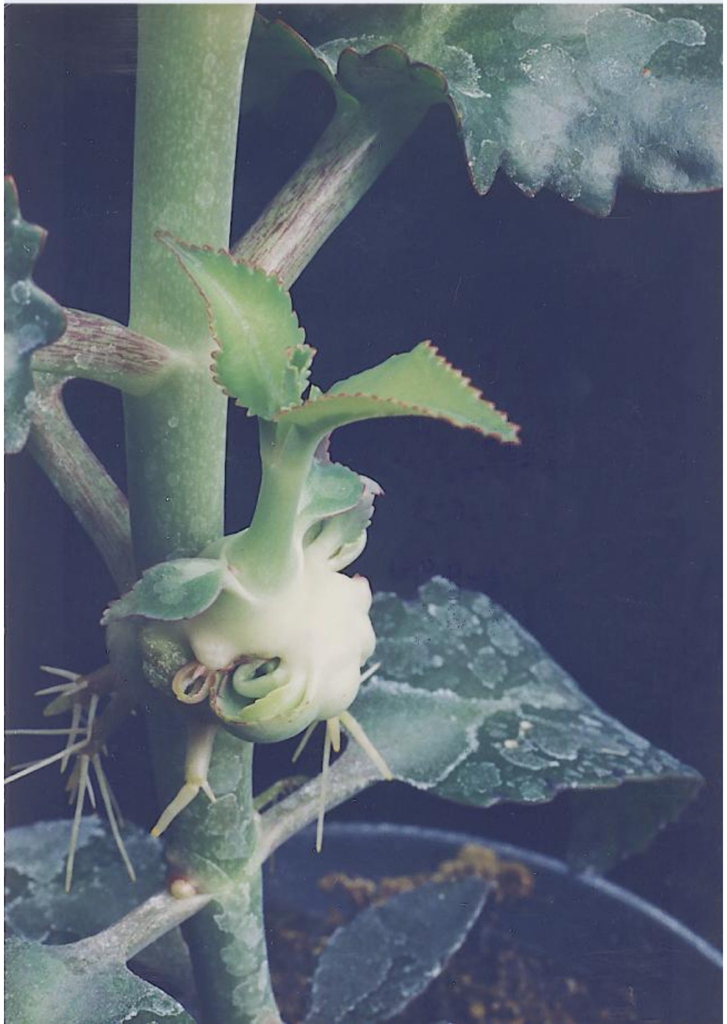
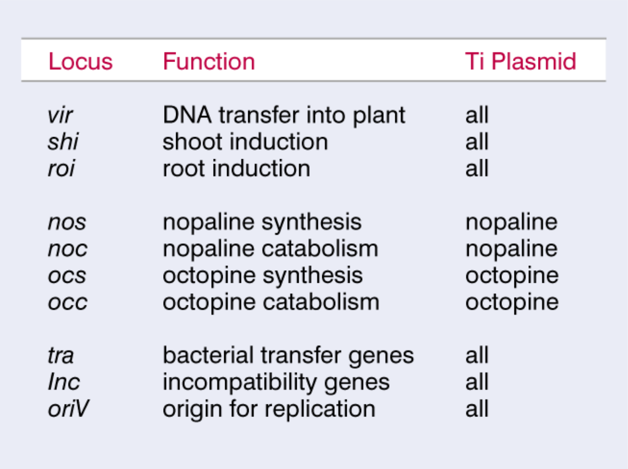
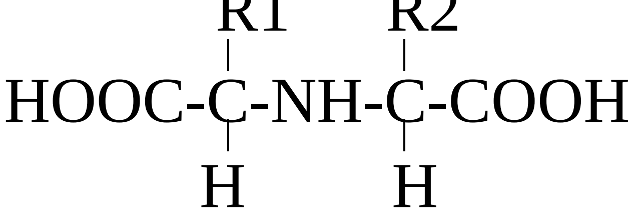


Figure 17.20 Ti plasmids carry genes involved in both plant and bacterial functions.



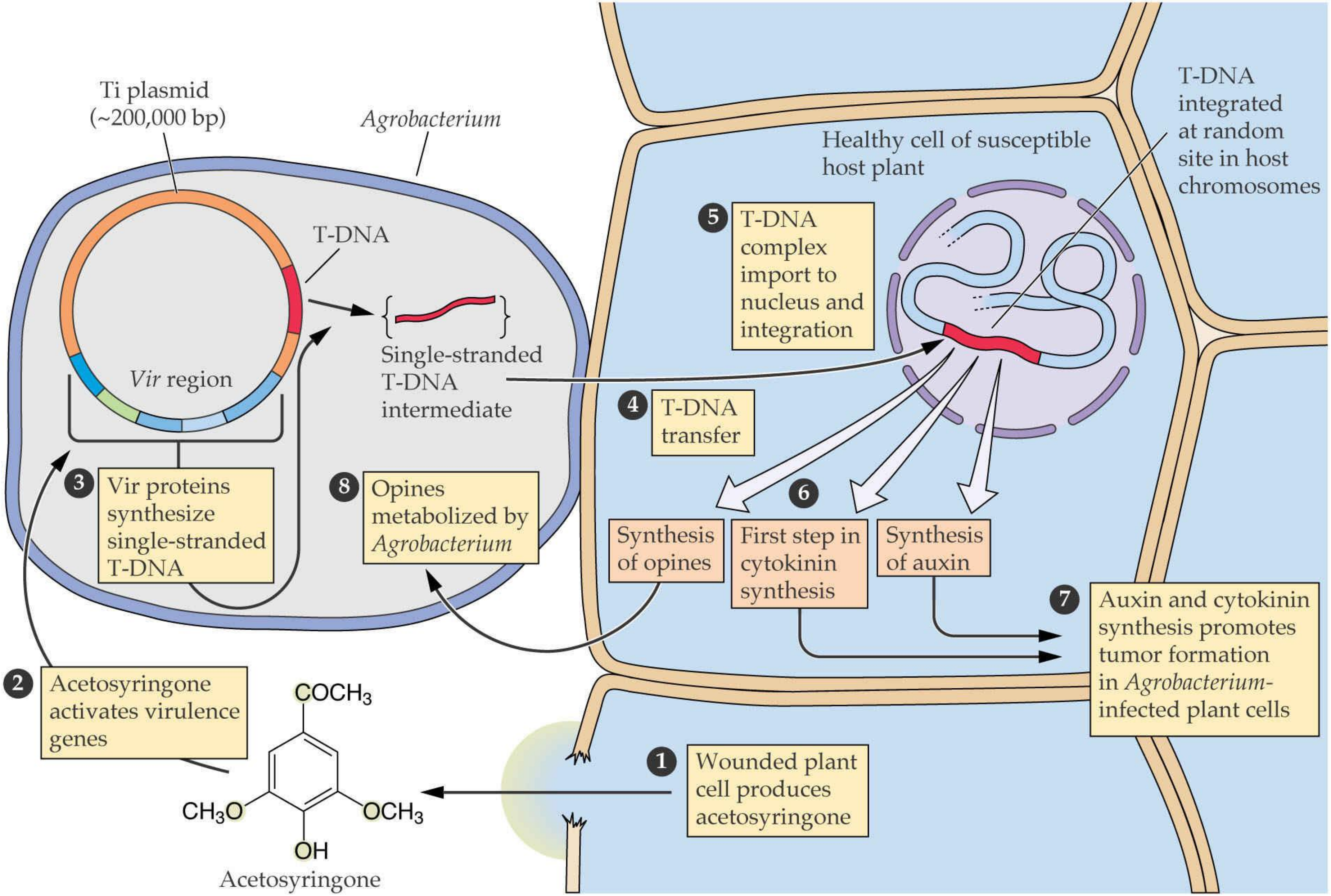
Locus	Function	Ti Plasmid
vir	DNA transfer into plant	all
shi	shoot induction	all
roi	root induction	all
nos	nopaline synthesis	nopaline
noc	nopaline catabolism	nopaline
ocs	octopine synthesis	octopine
occ	octopine catabolism	octopine
tra	bacterial transfer genes	all
Inc	incompatibility genes	all
oriV	origin for replication	all

Bacteria genetically engineer plants to control their differentiation (tumorigenic) and production of opines that can only be catabolized by the infecting Agrobacterium strain.



$$\mathrm{HOOC{-}}\underset{\underset{\mathrm{H}}{|}}{\overset{\overset{\mathrm{R1}}{|}}{\mathrm{C}}}\mathrm{{-}NH{-}}\underset{\underset{\mathrm{H}}{|}}{\overset{\overset{\mathrm{R2}}{|}}{\mathrm{C}}}\mathrm{{-}COOH}$$

T-DNA transfer, single strand invasion


Ti plasmid
(~200,000 bp)
Agrobacterium
T-DNA
Vir region
Single-stranded T-DNA intermediate
Healthy cell of susceptible host plant
T-DNA integrated at random site in host chromosomes
COCH$_3$
CH$_3$O
OCH$_3$
OH
Acetosyringone

1. Wounded plant cell produces acetosyringone
2. Acetosyringone activates virulence genes
3. Vir proteins synthesize single-stranded T-DNA
4. T-DNA transfer
5. T-DNA complex import to nucleus and integration
6. Synthesis of opines; First step in cytokinin synthesis; Synthesis of auxin
7. Auxin and cytokinin synthesis promotes tumor formation in *Agrobacterium*-infected plant cells
8. Opines metabolized by *Agrobacterium*

Transfer of T-DNA resembles bacterial conjugation

T-DNA is generated when a nick at the right boundary creates a primer for synthesis of a new DNA strand.

The preexisting single-strand that is displaced by the new synthesis is transferred to the plant cell nucleus.

Transfer is terminated when DNA synthesis reaches a nick at the left boundary.

The T-DNA is transferred as a complex of single-stranded DNA with the VirE2 single-strand binding protein.

The single stranded T-DNA is converted into double-stranded DNA and integrated into the plant genome.

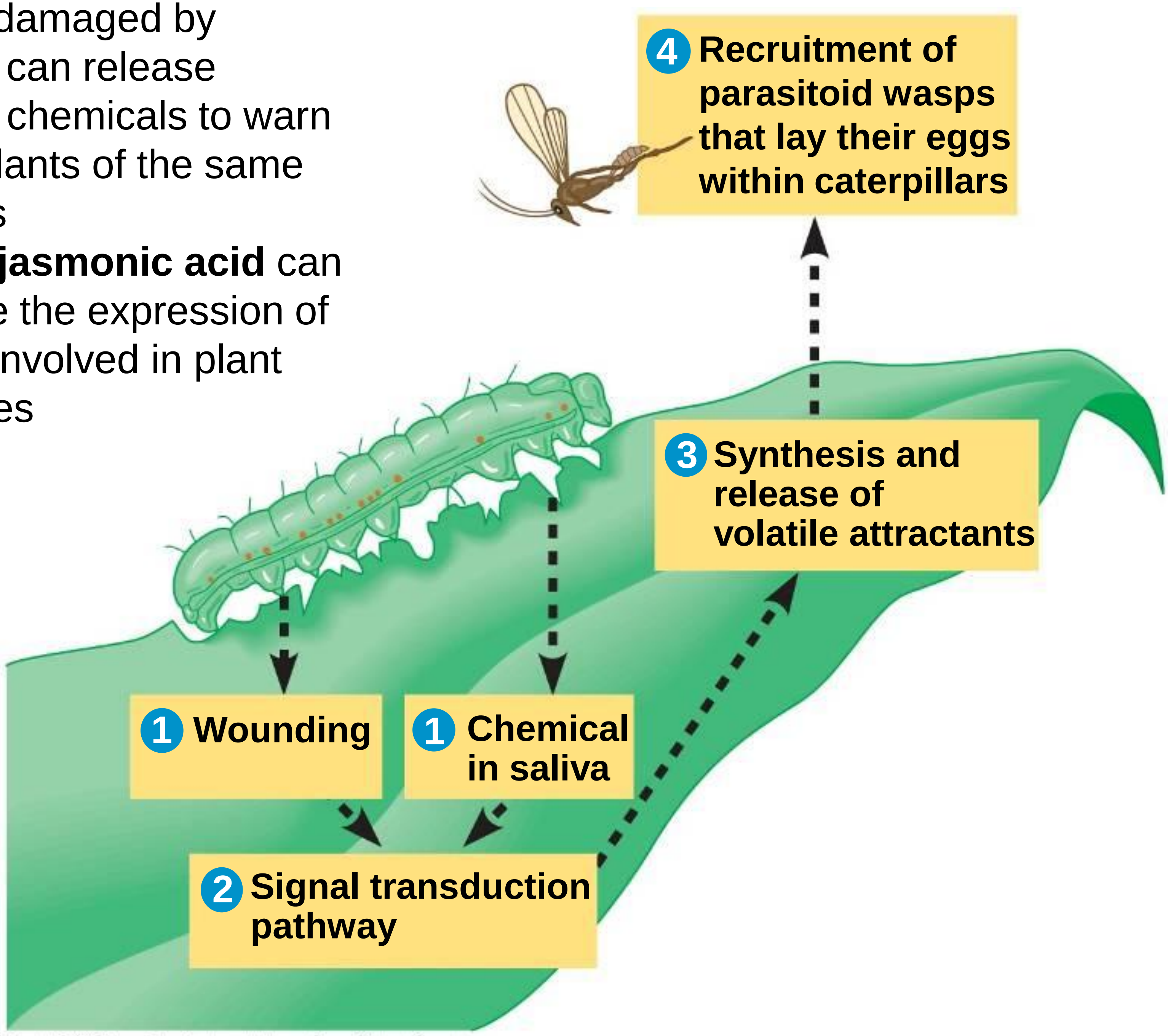
The mechanism of integration is not known. T-DNA can be used to transfer genes into a plant nucleus (transformation).

Defenses Against Herbivores

- Herbivory, animals eating plants, is a stress that plants face in any ecosystem
- Plants counter excessive herbivory with physical defenses such as thorns and chemical defenses such as distasteful or toxic compounds
- Some plants even “recruit” predatory animals that help defend against specific herbivores

Plants damaged by insects can release volatile chemicals to warn other plants of the same species

Methyljasmonic acid can activate the expression of genes involved in plant defenses



④ Recruitment of parasitoid wasps that lay their eggs within caterpillars

③ Synthesis and release of volatile attractants

① Wounding

① Chemical in saliva

② Signal transduction pathway

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